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Socioeconomic Status and Cancer in Alberta

by

Shirley M. Fincham ©

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Doctor of Philosophy

Department of Sociology

Edmonton, Alberta

Fall 1995

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UNIVERSITY OF ALBERTA

FACULTY OF GRADUATE STUDIES AND RESEARCH

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ABSTRACT

Successful intervention to alter behaviour, lifestyle and environment in cancer prevention requires that population subgroups at high risk are readily identifiable so that targeted programs can be developed for them. In the absence of population data concerning prevalence of direct disease determinants, socioeconomic status may be a useful indicator of these determinants and of risk for cancer. To supplement scientific knowledge on the relations between socioeconomic status and cancer incidence, the risk associated with various levels of SES was estimated for specific sites of cancer using case-control analysis. Three indices of SES were used: highest level of education, level of prestige associated with occupation (Pineo-Porter Socio-Economic Categories) and average annual, earned income associated with occupation, imputed from Statistics Canada census data. Cases were 7,385 Alberta men with cancer. For analysis at specific sites, controls were men with cancer at any site other than the focal site or with cancer of the lung. Odds ratios were estimated, stratified for age, cigarette smoking and alcohol consumption; multiple logistic regression was applied where stratified analysis produced statistically significant ORs. Tests for trend were also applied. All three indices were statistically significantly associated with risk for cancer at three sites. Risk for lip cancer decreased as all three indices increased. Risk for testicular cancer and non-melanotic skin cancer (NMSC) increased as income and education increased, but decreased as occupational prestige increased. Risk estimates significantly associated with income and education but not occupational prestige were: lung cancer (decreased with increases in education and income); malignant melanoma (increased positively with both); and prostatic cancer (decreased with both). Lung cancer risk also increased with cigarette

smoking, with statistically significant ORs of 1, 1.87 and 5.51 for never, light and heavy smokers respectively. Some statistically significant trends were demonstrated. Increments in income were associated with significant trends of decreasing risk for cancers of the lung and prostate and increasing risk for brain cancer. Risk for laryngeal cancer showed a significant trend to increase as occupational prestige decreased. These variables appeared to be indicators of the initiating or promoting factors which directly determine risk, cigarette smoking and exposure to ultraviolet light. This detailed examination of the association between education, income and occupational prestige and risk for cancer at specific sites has confirmed some previously reported findings with regard to income and education. The relationship between risk for cancer and prestige level of occupation has not been investigated before; these results have contributed new knowledge regarding this relationship. Occupational prestige appeared to be mostly a confounder, associated with work-place hazardous exposures, rather than with the social status of occupation. The analysis of these data support the use of income to identify subgroups at excess risk for lung cancer and for malignant melanoma. Public health interventions should focus on cigarette smoking prevention and reduction among low income earners to decrease lung cancer risk. A reduction in smoking behaviour would also reduce the incidence of laryngeal, bladder and stomach cancers. High income subgroups should be encouraged to avoid acute intermittent exposure to strong ultraviolet light and to reduce exposure to unfiltered indoor fluorescent lighting. The causes of brain and prostatic cancers are not sufficiently well characterized to permit planning interventions, but secondary prevention in the form of screening to detect disease early should be concentrated among those most susceptible, if limited resources preclude universal application of programs. The analysis also suggested several future lines of enquiry. Useful projects for the immediate future include analysis of the Alberta data by occupation and job exposure, controlled for income and education. Analysis by histological subtype in lung cancer and cutaneous malignant melanoma, controlled for these variables, may also prove informative.

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LIST OF SYMBOLS AND ABBREVIATIONS

SES - Socioeconomic Status

OR - Odds Ratio

95% CI - 95 Percent Confidence Interval

PYLL - Potential Years of Life Lost

SEER - Surveillance, Epidemiology and End Results Program

CHD - Coronary Heart Disease

MS - Multiple Sclerosis

ICD-9 - International Classification of Diseases - 9th Revision

ICD-O - International Classification of Diseases - Oncology

y - presence or absence of disease

x - vector of explanatory variables

p - response probability

Logit - logarithm of the ratio of frequencies of two different categorical outcomes

α - intercept parameter (alpha)

β - vector of slope parameters (beta)

g - function of mean of response variable

μ - mean

RERI - relative excess risk due to interaction

S - synergy index

AP* - proportion attributable to interaction

A - factor

B - factor

$\geq$ - greater than or equal to

$<$ - less than

NMSC - non - melanotic skin cancer

m - mean number of cigarettes per lifetime

R^2 - coefficient of determination

t - Student's t-test

CHAPTER I

INTRODUCTION

A. STATEMENT OF THE PROBLEM AND RELEVANCE

Cancer incidence and mortality are increasing throughout the industrialized world, as the population ages. The increasing social and financial impact of cancer must be reduced by preventing the disease. It is estimated that 60 to 70% of cancers are theoretically preventable[1], through changes in factors which can increase or decrease the risk of developing the disease. In order to develop successful intervention programs to alter behaviour and subsequent risk for cancer, it is necessary to be able to identify efficiently subgroups in the population who are at high risk. Detailed knowledge of the prevalence of the real determinants of disease in the population is not generally available; to collect population-wide data on risk and protective factors, such as tobacco use, diet, physical activity and exposure to sunshine, would be extremely difficult and expensive. A viable alternative may be to identify people at high risk through socioeconomic status (SES), which can be determined with relative ease, using demographic, residential, or sociometric measures, including the relative esteem accorded occupation by the community at large. SES has been correlated with a number of disorders, but consistent associations between SES and the development or retardation of cancer have not been demonstrated. If SES is to be used as a meaningful indicator for high risk groups, both consistent and site-specific associations must be observed. Therefore, to assess the usefulness of SES as an indicator of site-specific cancer risk and to increase scientific knowledge generally, cancer at selected sites was examined in relation to three separate measures of SES to detect possible associations.

B. OBJECTIVES

The specific objectives of this study were:

1. To establish whether the SES measures of education, earned, imputed income and occupational prestige are consistently associated with cancer at specific body sites among men with cancer in Alberta;
2. To establish which of these SES measures is the best predictor of risk for specific cancers;
3. To assess whether SES, operationalized by education, earned, imputed income or occupational prestige, is an indicator of direct disease determinants (that is, initiating or promoting factors which are highly correlated with SES), a potentially confounding indicator or an effect modifying indicator.

C. DATA SOURCES

A database exists in Alberta permitting an SES-based analysis of cancer morbidity, and in addition provided data for the control of possible confounders and an examination of interaction. The data base is from the Occupational Monitoring Project maintained by the Division of Epidemiology and Preventive Oncology, Alberta Cancer Board. It consists of lifestyle information including a lifetime occupational history, on approximately 7,000 male cancer cases registered in the province between 1983 and the present. Lifetime occupational history made it possible to estimate annual average income over a working career for each case. Occupational prestige rank was assigned to the occupation of the longest duration. Years of schooling was a variable already in the data base. The data base also includes women with cancer, but these were excluded because women have participated only since 1989, so that numbers are too small for any meaningful statistical analysis. An account of the author's involvement with the data base is attached in Appendix 1.

D. RATIONALE FOR SOCIOECONOMIC STATUS AS AN INDICATOR

SES was selected as a measure of social division because social class does not represent the realities of social stratification in modern industrial society. Class was traditionally conceptualized in economic terms. Karl Marx's original thesis was that the main class categories in modern capitalism involved the distinction between ownership and non-ownership of property. He distinguished two classes, the proletariat and the bourgeoisie, and predicted that class conflict was inevitable. Olin Wright expanded Marx's dichotomous class structure to account for people who did not fit into the original classes, such as semi-autonomous workers and small business owners. The basis for Wright's class differentiation was control, over investment and accumulation of wealth, over means of production, over labour power, and included intra-class exploitation through organizational assets, skills and credentials. His revised class scheme accounted for groups with mixed patterns of control, which better reflected the complexity of internal class differentiation in advanced capitalism. Also Wright's conceptualization did not include the inevitability of proletarian revolution. Alternatively, Max Weber stressed the class advantages resulting from the possession of knowledge and skills, and distinguished between class and status[2], although class was still the most important factor. J.H. Goldthorpe built upon Weber's theory and analyzed class on the basis of occupational function and employment status. Accounting for both the technical and social relations of production, in Goldthorpe's view class members are comparable in terms of sources and levels of income, degree of economic security and chance for economic advancement, and also in their location within the systems of authority and control governing the processes of production[2].

Succeeding neo-Marxist and neo-Weberian sociologists have expanded and revised the original class theories and approaches to class analysis to fit contemporary society. However, economic, technological and political changes, as well as changes in the labour force, have resulted in class no longer providing a suitable framework for the analysis of social stratification because the terminology does not correspond to the primary concerns of the contemporary labour force nor to modern organizational capacities. First, there are affluent workers in both the working class and the middle class; therefore, differences in attitudes and behaviours associated with economic advantage may be less apparent between classes. Second, technology has resulted in a decline of the number of people employed in manufacturing and exponential growth in white collar workers/low-level managers. It is not clear whether these last workers belong in the middle class, where they have been classified traditionally, or in the working class. Politically, collective action is increasingly organized on a non-class basis. Social movements such as feminism and the environmental lobby are not anchored in class, but operate across class strata. Class has declined in significance as a source of social identity; as production of material goods increased, access to and maintenance of consumption became major issues. Levels of consumption do not correspond consistently to class categories[3].

Because of the difficulties involved in defining class and applying class analysis in contemporary society, SES was selected as an indicator for attitudes, lifestyle, behaviours and environments, which may influence health and disease. Also, SES is more meaningful than social class because it lends equal weight to economic and social influences. The individual indices of SES were selected because of their availability, because their scales are hierarchically arranged and because they are assumed to represent different economic and social dimensions.

E. INDICES OF SES

Level of education is directly related to knowledge and the possession of skills, which confer a social advantage and improve life chances. Knowledge of the importance of the early detection of malignancy may prompt people with higher education to undergo screening for cancer more frequently, compared to people with less education. The knowledge that cigarette smoking is a major cause of lung cancer may persuade more people with higher education to stop smoking. There is evidence to show that cigarette smoking status (never smoked, ex-smoker, current smoker) varies strongly with educational attainment, with increased tobacco use as education level declines[4]. Better educated people are also more likely to have a strong sense of personal efficacy, to have an internal locus of control and to believe in personal initiative as a determinant of the future. All these factors tend to promote healthy behaviour and discourage risk taking[5].

Income is directly related to material wealth and life chances. Upper income groups may have better access to medical care than lower income groups. Early detection and treatment of pre-malignant conditions, such as colon polyps, may be protective in cancer at several sites[6]. People in lower income groups often do not have access to high quality dwellings and a clean external environment. Deteriorated respiratory health[7] and an increase in general mortality[8] have both been associated with increased environmental air pollution.

Occupational prestige level reflects social standing. Prestige has been defined as the esteem, respect or approval granted by an individual or the collectivity for performances or qualities they consider above average[9]. Prestige can be generated by above average performance in an area highly valued by the collectivity and perceived as contributing to the group. For example, in comparison with high-level management, professionals may have highly positive attitudes towards healthy behaviours, influenced by the social pressure of their occupational collective. It seems reasonable that their attitudes are likely to affect their personal behaviour, independent of the influence of education or income. Therefore, occupational prestige level should complement education and income as an additional measure of SES.

Although education, income and occupational prestige are assumed to be somewhat independent, there is also considerable overlap between the three indices. For example, level of education also governs job selection[5]. Better educated people are more likely to be employed at occupations which offer economic security, chances for economic advancement and a high degree of personal autonomy in performing their work. They are also less likely to encounter hazardous exposures in the course of their work. The result is that better-educated people are less stressed than those with little education and may be less subject to disease[5].

F. CURRENT KNOWLEDGE

Canadian Studies on SES

Very little work on SES as it relates to health and disease has been reported in Canada. An exception is the 1978 Canada Health Survey, which was analyzed with respect to SES. In this analysis, health status was defined by:

- the number of disability days in the two weeks prior to the survey
- the number of reported health problems
- medical history reflecting the prevalence of five specific chronic health problems
- the Bradburn scale reflecting psychoneurotic symptomology.

The analysis showed that indicators of health status improved as SES increased, with the exception of level of physical fitness, which showed the reverse. Income was consistently the best correlate of health status in this analysis and occupational status (with categories based on a combination of education or skill required plus income), was the least consistent[10].

A comprehensive review[11] of other Canadian studies has described inequalities in mortality from all causes among various SES groups. Included were a number of studies of occupational mortality, ecological studies based on neighbourhood income (estimates based on the percentage of people in income quintiles according to the previous census), studies of native Indian and Inuit mortality, infant death to birth linkage studies, mortality by income among contributors to the Canada Pension Plan and two studies on aging in Manitoba and Ontario[11].

More recently, changes in mortality by income in urban Canada between 1971 and 1986 were examined[3]. Income level was based on census data for neighbourhoods and only deaths occurring in [1]Census Metropolitan Areas[12] were included. In 1971, the difference in life expectancy at birth between the highest and lowest income quintiles was 6.3 years for men and 2.8 years for women. By 1986, these differences had decreased to 5.6 years for men and 1.8 years for women. In 1986, a major cause of death contributing to income inequalities in mortality was circulatory disease, accounting for 25% of the excess potential years of life lost (PYLL) before the age of 75 and attributable to income quintile differences. Accidents, poisoning and violence combined and malignant neoplasms accounted respectively for about 17% and 15% of PYLL due to income differences. Other income inequality-related causes contributed less than 7%. Only about 36% of PYLL was not income inequality-related. In terms of age-standardized mortality rates for all ages, certain causes of death showed increased mortality in urban areas with greater income inequality, especially for males. These were lung cancer, suicide, metabolic diseases other than diabetes and ill-defined conditions[13].

---

[1] Census Metropolitan Areas consist of census subdivisions which fall completely or partly inside an urbanized core, in which at least 50% of the employed labour force living in the subdivision also work in the urbanized core and in which at least 25% of the employed labour force working in the census subdivision also lives in the urbanized core.

Differences in health behaviours were also found to be related to SES in Canada's Health Promotion Survey[14]. Lower-income, less-educated Canadians were more likely to rate their health as poor, compared to high income, better-educated people. However, higher-income Canadians were more likely to drink heavily, to drink and drive, to use marijuana and report high levels of stress. Lower-income people used more tranquillizers, got less exercise, were less knowledgeable about first aid and cardiopulmonary resuscitation. They were less likely to have home safety devices, to use seatbelts and were more likely to be exposed to sidestream smoke in the workplace and to have friends who smoke. Compared to employed Canadians, the unemployed were less likely to report excellent health and reported consistently poorer health habits. The only exceptions were drinking and driving and prevalence of overweight, both characteristics more common among the employed.

Cancer in Canada and SES

There has also been one report published on the association between SES and a specific site of cancer. A small increase in risk of malignant melanoma with increased SES was reported in a case-control study from British Columbia. The increased risk was greatly reduced after control for phenotypic attributes (skin and hair colour, freckling, skin reaction to sunlight, ethnicity)[15]. A similar positive association between risk for melanoma and SES was found in Washington State, but only for males between 30 and 69. At 70 and over, the trend was reversed. The investigators suggested that cumulative sun exposure may be the operative factor and that the poor elderly have more cumulative lifetime exposure[16].

To date, there have been no other studies in Canada of the impact of SES on the incidence of cancer at specific sites, controlling for the effects of possible confounders. The Alberta data analysis partially corrects this and may stimulate additional study in other regions of the country.

Cancer in Other Regions and SES

Compared to studies of SES and other diseases, work on the relationships between SES and cancer is also sparse in other countries and the findings are inconsistent[17-34].

The majority of recent studies have occurred in countries with large and readily identifiable minority groups, in which it has been difficult to separate the possible effects of SES from those of other, ethnic-specific factors. For example, compared to Anglo women in the United States, Hispanic women experience less breast, colon and lung cancer[17]. Hispanics generally hold jobs of lower status, have lower incomes and less education than the Anglo population. There are differences between the two groups in diet which are related to ethnicity rather than to income. Low-income Hispanics in a nutrition survey[17] reported low meat consumption and high legume and milk consumption, whereas low-income Anglos reported high cereal and starch consumption. Hispanics also have the highest fertility of any ethnic group in the United States, an ethnicity-related factor which may be protective in breast cancer. It is not clear whether these differences in cancer occurrence are due to factors related to SES, to ethnicity-related differences or to genetic predisposition.

Another example concerns the difference in cancer rates between blacks and whites in the United States, only some of which might be related to SES. Where data from the U.S. National Cancer Institutes' SEER program were adjusted for three indicators of SES (census district median income, education and population density), risks for cancer at all sites and for lung cancer were reversed. Lung cancer declined from a significantly higher rate among blacks before SES adjustment to a significantly lower rate compared to whites. Adjustment for SES did not affect the excess risk in blacks for stomach and prostate cancers, suggesting these may be associated with unknown cultural or genetic traits unrelated to SES, which should be investigated[18] (please see Limitations of Published Studies section on page 19). Another example concerns an interesting cross-over in female breast cancer rates between black and white women in the United States, which might be due to variation in reproductive habits with SES. Among women over the age of 40, white women have higher rates compared to black women; among women under 40, the reverse is true[19]. Risk for breast cancer should be consistently higher among white women, because the prevalence of risk factors is higher among them. Compared to black women, white women tend to marry and have children later and to have fewer children altogether. It is suggested that the excess rate among black women under 40 may be a result of social determinants such as lower SES, which promote the prevalence of abortion at an early age, prior to a first full-term pregnancy. Teen-age pregnancy is also more common among blacks; the proportion having a first full-term pregnancy by age 18 has been two to three times as high as among whites[20]. Exposure to oral contraceptives is also higher among black women. These reproductive patterns increase the vulnerability of breast tissue, by increasing hormonally the number of undifferentiated cells and susceptibility to exogenous carcinogens. The result would be excess pre-menopausal breast cancer rates, compared to those expected, among black women.

A number of non-SES related factors have been proposed for the excess prostatic cancer rates in blacks[8]. An hormonal mechanism may be responsible, as testosterone is necessary for the prostate gland's growth and functioning. Behaviourial factors such as sexual habits and diet may affect hormones, leading to greater risk. Occupational exposure to cadmium and urban living have been implicated, as well as dietary constituents not associated with hormone functioning, such as zinc, vitamins A and C. Further effort is needed to explicate the effects of SES, genetic predispositions and ethnicity-related lifestyle factors[21].

Mortality studies have contributed some evidence relating cancer risk to SES. In the United Kingdom, calculation of standardized mortality ratios indicated that mortality from all cancer sites together increased from 75 in Social Class I (professional and similar occupations) to 131 in Social Class V. Mortality was also higher for the manual versus non-manual component of Social Class III[22]. Social class differences in cancer mortality in New Zealand were generally consistent with the United Kingdom results, showing excess risk in the lower social classes for cancers of the liver, larynx, lung, buccal cavity and stomach, and excess risks for multiple myeloma, malignant melanoma and lymphatic leukaemia in the upper social classes[23].

On the other hand, an examination of mortality data in the Alameda County Study, a prospective cohort study in the United States, showed no differences in mortality by

SES for any specific cause of death, including cancer[24]. In contrast to these reports, a correlational study of cancer, as determined from histopathology reports, and socioeconomic and demographic indicators in Brazil found that lung, laryngeal and colon cancer were all highly positively correlated with indicators of affluence[25]. Recent incidence data from Finland were analyzed by education and a combined measure of a number of background variables describing a municipality's social welfare (average income per inhabitant, percentage of population belonging to two highest social classes, percentage of educated persons and percentage of persons in industry). The results showed high cancer rates in low SES groups for oesophagus and stomach, but high rates in high SES groups for colon, rectum, prostate, testis, kidney, malignant melanoma, female breast and corpus uteri[26,27]. Similarly, risk of neuroblastoma was higher among children whose mothers were college graduates and whose fathers were employed as professionals during the pregnancy in Tennessee[28]. However, a number of maternal factors were associated with excess risk in this study. These were maternal use of diuretics for high blood pressure, use of tranquillizers, analgesics, pain relievers and cigarettes. The association between drug use and education is somewhat confusing. In the Canadian Health Promotion survey[14], higher tranquillizer use was associated with low income. While more people at low SES levels smoke, among those who do smoke, cigarette consumption is lower at this level compared to the high SES level.

Examination of incidence and mortality studies from a number of other countries suggests that the most consistent findings are for excess rates of oesophageal[29], stomach[30,31], lung[32] and cervical[1] cancer in low SES groups and excess female breast cancer[33,34] in high SES groups. However, even for these sites the data are not altogether consistent and for other sites, more evidence is needed before clear trends emerge.

In addition to incidence and mortality studies on all sites, a few reports have been published on the association between SES and specific sites of cancer. Increased risk for testicular cancer has been related to upper social classes, professional occupations and high income in several studies[35]. Increased risk for oropharyngeal cancer has been correlated with one measure of SES (percent of working life spent in employment) in an inverse relationship[36]. In the latter study, there was no association between oropharyngeal cancer and two other measures of SES, education and occupational status (defined in terms of degree of prestige as perceived by the public).

Finally, Falk and colleagues reported white collar workers were at higher risk of pancreatic cancer in Louisiana. This finding was inconsistent with other studies they reviewed, which reported either no association with SES or excesses in low SES groups[37].

Other Diseases and SES

In general, current studies have demonstrated consistently that low SES is associated with lower life expectancy, higher mortality from all causes of death combined, higher rates of infant and prenatal mortality and higher rates of a number of major mental disorders[38]. For example, rates of low birth weight are similar among poor black women, poor white women and non-poor black women. Rates in these groups are about twice as high as rates of low birth weight among non-poor white women[39]. This suggests that in addition to poverty, other factors are operating differentially. The

American epidemiologist Nancy Krieger has hypothesized that the experience of racial oppression may be responsible for low birth weights and generally poorer health among blacks, through limited access to health care. Even at the same economic level, blacks and whites receive differential care. A second factor, which requires study, is possible physiological change which may be associated with response to acts of discrimination.

Among the chronic diseases, coronary heart disease (CHD) used to be more prevalent among affluent males[40]. In the early 1900s, William Osler observed that angina pectoris was an "affection of the better classes". Somewhat later, mortality analysis by occupation showed a striking social class gradient in CHD mortality in Britain for the period 1930-32, increasing from lower to upper classes[41]. Data from American studies in the 1930s and 1940s largely confirmed these findings[42]. A shift in social class distribution began about 1950, when mortality was concentrated in the lowest social class among men under the age of 45 in both Britain and the United States. The shift in both coronary mortality and morbidity continued in the 1960s, as was apparent in studies of rich and poor states in the U.S., from the National Health Interview Survey and from studies of selected metropolitan areas[43].

A decline in CHD mortality also occurred in Britain, but later than in the U.S. Beginning about 1979, CHD mortality decreased about 15% among men in the non-manual occupational class, although it increased at the same time by 1% among manual workers. Higher rates of heart disease in lower social classes are also being reported from Australia, New Zealand, Finland, Norway, France and Sweden[44].

The change in disease pattern is partly a result of the change of risk factor prevalence, which has decreased more in higher SES groups; it has been shown that obesity[45], smoking[4,46-48], stress and elevated blood pressure[40], all of which are associated with CHD incidence and mortality, are more prevalent among low income groups.

Table 1.1[49]
Trends in the Prevalence of Smoking Among Adults

CHD Mortality	Country	Time Period		Percent of Current Smokers	
				Earlier Period	Later Period
38% to 18% Decline	U.S.	1976-1986	Men	42	35
			Women	33	30
	Canada	1972-1983	Men	47	31
			Women	32	28
	Japan	1972-1980	Men	78	70
			Women	16	14
	Netherlands	1970-1982	Men	75	41
			Women	42	33
13%to 0% Decline	U.K.	1970-1984	Men	68	36
			Women	44	32
	Denmark	1972-1980	Men	68	57
			Women	49	44
	Norway	1970-1980	Men	56	46
			Women	37	39
	Italy	1965-1980	Men	60	54
			Women	13	17
11% to 60% Increase	Czechoslovakia	1974-1984	Men	61	57
			Women	29	14
	Spain	1981-1986	Men	58	58
			Women	20	23

However, the relationship between risk factor prevalence and disease is not as straightforward as might be expected. Changes in smoking have occurred in countries with dramatic declines in CHD mortality. However, changes in smoking have also occurred in countries with moderate or no CHD mortality declines and in those with increases in CHD mortality. Table 1.1 shows some changes in CHD mortality and corresponding estimates of current smokers for a number of countries. The current smoker percentages given are associated with the first and last years respectively of the listed time periods. In general however, decreases in smoking, particularly among men, are paralleled by decreases in CHD mortality.

Globally there are also dietary differences between SES groups, although total fat intake is about equal. In higher income groups the consumption of fruits and vegetables is strikingly higher[44] and vitamin C may be protective in CHD. In two studies of plasma micronutrients, lower levels of plasma vitamins C and E were associated with high risks for angina pectoris[50] and CHD[51]. Also, in a double blind clinical trial with placebo control, administration of ascorbic acid for six weeks lowered systolic blood pressure and pulse pressure (the difference between systolic and diastolic blood pressures), in a group of borderline hypertensives[52].

Disadvantageous factors operating early in life among the poor also influence disease risk in middle age. An analysis of regional variations in CHD mortality in Norway showed a strong association between CHD and poverty at birth and in infancy[53]. A British study showed that short height predicted CHD mortality independent of grade of employment; although height is primarily genetically determined, it can be influenced by early environment. Increased intake of macronutrients during early growth is associated both with increased stature and with lean body mass[54].

Diabetes mellitus has also been shown to be related to SES. Non-insulin dependent diabetes mellitus is associated with obesity, which occurs more frequently in lower SES groups. Studies of obesity have almost uniformly demonstrated the importance of SES among women and to a lesser extent, among men. In the United States, the percentage of overweight women was highest in the poverty group[55]. SES may be a proxy for a more direct variable, such as maternal age - a number of studies of diabetes mellitus have demonstrated that risk increases with maternal age[56]. Therefore, it is somewhat surprising that non-insulin dependent diabetes mellitus occurs more commonly in upper SES groups. It has been suggested that these people have more frequent medical testing and that the disease is underdiagnosed in lower SES groups. On the other hand, malnutrition-related diabetes mellitus is associated with poverty. It occurs primarily in tropical countries and is a result of malnutrition, particularly protein deficits[57].

The development of multiple sclerosis (MS) has also been related to SES, but the evidence is not convincing. In Wales, MS was more prevalent among middle aged, Caucasian women, born in England rather than Wales and occupying flats in affluent communities with superior amenities where rates of owner occupation are high. MS was least common among young, single, Welsh-born or non-Caucasian males, living in rented accommodation in less affluent communities, with a high population density[58]. In Scotland, two thirds of MS cases belonged to social classes I to III. This preponderance in the higher classes was significant when compared to the distribution in the general population[59]. On the other hand, MS in a French study appeared to be associated more with occupational exposures than with SES. High rates were observed among farmers, physicians, nurses and hairdressers[60]. Two Canadian case control studies failed to find differences between patients and population controls in several SES measures. Alberta MS cases did not differ from a hospital control group in level of education, current occupation, adequacy of income at onset or occupation before onset[61]. Ontario cases and controls were similar in educational level, as well as marital status and religion[62].

One index of SES has been considered in Alzheimer's disease. Although the strongest evidence currently implicates genetics as the major causal factor[63,64], there is a suggestion that risk for this form of early onset dementia may be associated with education. In the United States, Alzheimer's is more prevalent among blacks than among whites[65]. A recent conference summary reported that population studies in many parts of the world suggest lack of education may be a factor, particularly early education. Neocortical synapses are known to be degraded in Alzheimer's and the current hypothesis suggests that early education helps provide a reserve of these synapses, which delays the onset of dementia[66].

Limitations of Published Studies

Studies published to date have a number of limitations which contribute to the inconsistency of results. First, the conceptual basis for SES and the health/disease relationship is somewhat obscure. It is not clear whether the conditions of a low SES life initiate or promote disease (social causation hypothesis) or whether incapacity due to already developed disease or to predisposing conditions produces downward mobility (social selection or drift hypothesis). It is not possible to resolve the question of which is cause and which consequence without analysis of longitudinal data. However, it has been argued that education may be an indicator of social causation[10], as highest level of education is attained fairly early in life and is therefore more likely to affect health than is health to limit educational attainment. Income, on the other hand, can be affected by ill health and may more closely reflect the social selection hypothesis. Whether measured by level of prestige, education or income, occupational status is primarily a function of both education and income and may be an indicator of both hypotheses.

Second, in addition to the problem of distinguishing between SES and genetic or ethnicity-related factors as previously discussed, there is a lack of standardization in the specific measures of SES used and it is not clear that all the individual measures reflect the same phenomenon[38]. Some of the studies also use social class rather than SES, increasing the difficulty with generalizability of results.

Liberatos and her colleagues[38] reviewed the most common indicators of SES. Occupation is commonly used and is considered to be related to health in a number of ways. Health may be affected by workplace exposures to physically noxious or psychologically stressful environments, degree of intrinsic and extrinsic rewards (including degree of job prestige), job security, personal control in the environment and the ability to obtain good housing. Education is considered to be related to health outcomes through its influence on lifestyle behaviours, problem-solving capacity and values, such as the importance of preventive health behaviours. Income, the third most commonly used measure, overlaps both occupation and education and is considered to provide access to good housing, less exposure to noxious environments, good diet, good working conditions and more social amenities. A partial list of other indicators of SES includes population density, standard of living and quality of neighbourhood measures, such as proportion of people below the poverty line, with poor education, proportion of renters versus homeowners, costs of housing and proportion unemployed.[18,25,26]

Most investigators[18,38] advise against the use of composite indices of SES for a variety of reasons. Multiple single indicators provide more information and more flexibility and can be combined if it seems useful. Current multivariate analysis methods are capable of controlling simultaneously a large number of separate measures. Finally, the use of composite indices may obscure important differences of theoretical or practical significance.

In addition to the Greenberg report[36] on oropharyngeal cancer being associated with percentage of working life spent in employment but not associated with education or prestige level of occupation, there has been considerable variation in relations between individual measures and other outcome variables, including physician visits, depression, pain, disability days, number of health problems and medical history[10,38]. For example, Hollingshead and Redlick[67] investigated downward mobility in schizophrenia using a composite SES measure heavily weighted by education. They found no evidence of downward mobility, perhaps because schizophrenia develops in late adolescence or early adulthood, often after education is complete. Other studies, using occupation-based SES measures, found clear evidence of downward mobility[68,70]. Selection of an inappropriate SES indicator can lead to misclassification and possibly dilution of effects.

Another problem with SES studies is that the various indicators may change over time, thus producing discrepant results using the same indicator. For example, in Canada between 1961 and 1986, the number of university graduates rose 432%[71]. However, the economy has not kept pace with education and as a result, a high level of education may be associated with a relatively lower income in 1986 than was the case in 1961.

A final limitation of the majority of the studies on SES and health and disease is the inability to control for possible confounders. Death certificates and cancer incidence data from registries do not contain data on lifestyle habits such as smoking and alcohol, both of which vary with education, social class and income, but not always consistently[72-75]. The occupation recorded may be the most recent one held, rather than the usual occupation of longest duration, or it may be mis-recorded as one of greater prestige than was actually the case[76]. In both instances, occupational misclassification might result in bias which would be difficult to quantify.

This study was designed to overcome some of these limitations. The use of three separate indicators provided data which were intended to contribute to clarification of theoretical issues of social selection or causation surrounding the SES-disease relationship. Prevalence of the determinants of cancer risk varies over the levels of the three hierarchical SES indices. Finally, control for the most important possible confounders (that is, age, smoking history and alcohol consumption) assisted in the assessment of SES as an indicator of a disease determinant, a confounder or an effect modifier.

CHAPTER II

METHODS

A. SUBJECTS

Subjects were a sub-sample of men with cancer from the Occupational Monitoring database described in the introduction. This database was established to remedy the deficiencies of analyzing possible associations between occupation and cancer, using occupational data derived from death certificates, or using limited cancer registry data. The Alberta Cancer Registry recorded occupation between 1978 and 1989, but the data were of poor quality and occupation was missing for about half of the male registrants. Occupational information has since been deleted entirely from the Registry.

Therefore, the Division of Epidemiology and Preventive Oncology of the Alberta Cancer Board, with financial assistance from Alberta Occupational Health and Safety and the Alberta Workers' Compensation Board, began a project in 1983 with two objectives: 1) to scrutinize continually cancers known to be related to occupational exposures, so that better control of these hazards can be exercised in the province, and 2) to examine the distributions of occupations in all types of cancer, controlling for known confounding factors, so that new hazards can be quickly detected and appropriate hypotheses can be formulated and tested concerning these hazards.

For this analysis, included from the main sample were males diagnosed with a first primary malignant neoplasm between 1983 and 1990 and Alberta residents at diagnosis. Only cancer cases with a diagnosis confirmed by histology or cytology were included.

The main data base is restricted to cases who are alive at diagnosis both to reduce project costs and because surrogate data on occupational history are of questionable value[77,78].

The database was restricted to those aged 25 to 74 at diagnosis because there is some evidence that occupational cancers occur earlier in life than those associated with other factors and that under the age of 24, occupational exposure would be of short duration and therefore would not yet have caused an effect[79]. In situ and borderline malignancies were excluded because of uncertainty with regard to the natural history, correct diagnosis and tumour progression prevents meaningful risk factor analysis. People participating in other studies were excluded. Recently, the excluded group has included non-melanotic skin cancer cases in Southern Alberta and thyroid, testicular, prostatic and nasopharyngeal/sinonasal cancer cases. Between November 1990 and March 1991, colon cancers were excluded, as these were involved in a case-control study of risk factors in diseases of the large intestine. Also excluded were cases for whom no address or telephone number was found and those where the physician advised against contact (4% of all cases), as no data were available for these people. Details of exclusions are shown in Table 2.1.

This analysis of SES was limited to males because women have only recently been included in the occupational monitoring database and numbers are still too small to permit meaningful statistical analysis.

B. IDENTIFICATION OF CASES

Cases in the main database from which this sub-sample was selected were identified through the Alberta Cancer Registry, which is operated by the Division of Epidemiology and Preventive Oncology of the Alberta Cancer Board. Data on patients with cancer in Alberta have been collected since the early 1940s, when a file-card data collection system was initiated to assist in the administration of payments for services rendered to cancer patients. From this modest beginning, the Registry has evolved to become a controlled mechanism for the collection of clinically relevant information on all persons diagnosed with cancer in Alberta.

Table 2.1
Exclusions from Occupational Monitoring

Reason for Exclusion	Site	Years	Approximate Number of Cases
Other Studies	Thyroid	87-88	65
	Testis	83-85	148
	Prostate	83	582
	Non-Melanotic Skin (Southern Alberta Only)	85-88	3000
	Nasopharynx/ Nasal Cavity	83-87	138
	Colon	90-91	400
In Situ		83-88	916
Physician Refusal		83-91	745

Since 1968 it has been mandatory that all private and hospital laboratories send any pathology reports that deal with malignancies to the Registry. After receiving the pathology report, a registration form is sent to the attending physician, requesting information about the patient and tumour. For patients who attend a facility operated by the Alberta Cancer Board, Registry coders abstract data directly from clinical charts. Data on patients who are not referred to a cancer facility and on those who fail to report are obtained from relevant documents that are sent to the Registry. The main sources of information for the Registry are pathology reports, hospital discharge summaries, operating room reports and death certificates.

C. MEASURES OF SOCIOECONOMIC STATUS

Education

To assess the effects of education on cancer four levels were selected and analyzed by cancer site:

1. Completed elementary school only (1-9 years of school).
2. Completed secondary school only (10-13 years of school).
3. Any post-secondary education except 3 or more years of university.
4. University (≥ 3 years of university or college).

The third category included technical, trade, vocational and non-degree college training. A final category in the questionnaire, that of "Other", was included in the third group where possible, but otherwise was not analyzed.

Occupational Status

The 1971 Pineo-Porter socio-economic categories for occupational status were used, because these were based solely on occupational prestige[80]. Although other measures of occupational status include either or both income and education, the Pineo-Porter classification system was selected on the assumption that the social standing of an occupation may reflect a somewhat different dimension than do income and education.

The Pineo-Porter scale[80] includes 16 categories of occupational prestige (defined in detail in appendix II):

01	Self-employed professionals
02	Employed professionals
03	High-level management
04	Semi-professionals
05	Technicians
06	Middle management
07	Supervisors
08	Foremen
09	Skilled clerical-sales-service
10	Skilled crafts-trades
11	Farmers
12	Semiskilled clerical-sales-service
13	Semiskilled manual
14	Unskilled clerical-sales-service
15	Unskilled manual
16	Farm labourers

Prestige was established using the social standing ratings associated with 204 occupational titles by a representative population sample of 793 persons in 1961 in Canada. The scale was revised to conform to occupational coding changes in 1971[81], then to be compatible with the 1980 four-digit occupational codes[82] used in the Occupational Monitoring data base.

Income

Earned income by occupation data are available for the census years 1931[83], 1936[84], 1941[85], 1961[86], 1971[87], 1981[88] and 1986[88], from Statistics Canada. Lifetime average annual earned income for each individual was calculated as follows:

1. Four-digit occupational codes were entered into the data base, paired with the appropriate census years and average annual incomes.
2. For years in which no income by occupation data were available, income was estimated by assigning the incomes of a specific census year to the other years in the interval surrounding that census year. This was done arbitrarily, because the intervals between census years were not regular. Census year incomes were assigned as follows:

   1931 average annual income to 1929-1933
   1936 average annual income to 1934-1938
   1941 average annual income to 1939-1956
   1961 average annual income to 1957-1966
   1971 average annual income to 1967-1976
   1981 average annual income to 1977-1983
   1986 average annual income to 1984-1991

3. Lifetime average annual earned income was calculated:

$$\frac{\text{sum of all average annual incomes over employment duration}}{\text{number of census years over duration in which incomes were available}}$$

This method for ascertaining income was selected because it was readily calculated from the occupational history data. The method has the added advantage of taking into account income over a working life, rather than only current income. The induction period for most cancers is in excess of ten years and current income may not reflect earlier income, which would be more important in relation to the development of cancer.

Possible effects on earned, imputed lifetime income of age at starting employment were controlled for by including age in stratified analysis and in multiple logistic regression analysis. In the Occupational Monitoring Project, respondents are not asked about income because this question was considered to be too intrusive and an unnecessary invasion of privacy.

D. DATA SOURCES

Data on lifetime occupational history, usual occupation, occupational exposure to hazardous materials, education, family history of cancer, tobacco use, alcohol consumption and residential history were abstracted from the Occupational Monitoring database for men who recorded their education and for whom a usual occupation could be established. Usual occupation was defined as the occupation worked at for the longest duration. Where two occupations had been held for the same length of time, the most recent was arbitrarily selected as the usual one.

Data were originally collected from eligible new registrants with cancer using a mailed, self-administered questionnaire (copy attached in Appendix III), with follow-up to encourage response and supplement incomplete information.

Tables 2.2 to 2.4 show the response rate, reasons for refusing to participate and the improvement which occurred with follow-up for the main database between March 1989 and June 1991. In terms of people with whom contact was made response was quite acceptable (81%). Twelve percent of the eligible sample could not be contacted for the reasons listed in table 2.2

Data Coding and Entry

Cancer site coding was according to the 1990 ICD-O, Second Edition[89], rules. Previous disease codes in ICD-9[90] were converted to ICD-O. Specific codes and sites grouped together for analysis are included in Appendix IV. For the other variables in the questionnaire conventional coding methods were used. A randomly selected 10% sample of coded questionnaires was reviewed by a second coder to assess accuracy. A random 25% selection of questionnaires was entered twice for the same reason. The error rate was less than 1% for both coding and entry. All income data were entered twice to ensure accuracy.

E. SAMPLE SIZE CONSIDERATIONS

The statistical power to detect an effect which is associated with a number of sample sizes is shown in Table 2.5[91]. In this study, the approximate distribution over 17 cancer sites ranged in frequency from a low of 0 to a high of 772 cases, with a minimum case/control ratio of 1 to 24. Given such a favourable ratio, with only 25 cases there was about a 50% chance of detecting an OR as low as 3 even when the prevalence of the risk factor in the general population was about 5%, with $\alpha = 0.05$ and a two-sided test. The problem of multiple comparisons and statistical significance will be taken into account in the interpretation of the results.

Occupational Monitoring
March 1, 1989 - June 30, 1991

Table 2.2
Response Rate

	#	%
Eligible Identified Cases	4289	
Physician Refusals	166	4
Returned Address Unknown	76	2
Returned - Case Deceased	253	6
Moved Out of Province	33	<1
Total Not Contacted	528	12
Refusals	307	7
Questionnaires Returned	3055	
Questionnaires Returned ÷ by All Eligible Cases		71
Questionnaires Returned ÷ by All Contacted Cases		81

Table 2.3
Reasons for Refusing to Participate

	#	%
Total Refusals	307	
Too Ill	39	13
Language Difficulty	12	4
No Reason Given	256	83

Table 2.4
Improvement with Follow-up

	#	%
Total Returned	3055	
Returned Without Follow-up	2243	73
Returned With First Follow-up	330	11
Returned With Second Follow-up	482	16
Total Returned With Follow-up	812	27

Unfortunately, the power to detect significant differences from the risk estimate predicted by the null hypothesis decreased when cell numbers were below 25 cases, severely limiting the reliability of the ORs for rare cancer sites in this study. However, for most sites, the frequency of cases was over 25.

Although it is not known what the distribution of income, education and occupation is across cancer sites, there are published data on the prevalence of these factors in the Canadian population. About 20% of Canadians have university or community college education, about 20% of Canadian family incomes were over $53,400 or under $17,834 in 1985[92] and occupational category distribution in Alberta in 1991 ranged from a low of 1% for Material Handling and Other Crafts to 16% for Managerial and Professional occupations [93], so power for most cancer sites and risk factors in this study was sufficient to justify analysis.

Table 2.5
Power to Detect an Effect
($\alpha=0.05$, Two-Sided Test, Control/Case Ratio = 24:1)

Number of Cases	Odds Ratio	Prevalence of a Factor in the Population					
		0.05	0.1	0.2	0.3	0.4	0.5
10	2	0.09	0.13	0.17	0.19	0.19	0.18
	3	0.22	0.33	0.41	0.42	0.39	0.34
25	2	0.17	0.26	0.37	0.40	0.40	0.37
	3	0.47	0.67	0.79	0.79	0.76	0.69
50	2	0.29	0.46	0.63	0.68	0.68	0.64
	3	0.70	0.91	0.97	0.98	0.96	0.93
100	2	0.52	0.75	0.90	0.93	0.93	0.90
	3	0.96	1.00	1.00	1.00	1.00	1.00
300	2	0.93	1.00	1.00	1.00	1.00	1.00
	3	1.00	1.00	1.00	1.00	1.00	1.00
500	2	..	..	..	..	..	..
	3						

CHAPTER III

DATA ANALYSIS

A. CONTROLS

The use of cancer controls is common in occupational and registry-based studies and there are a number of advantages to using these rather than population controls. Selection bias is reduced because other registered cancers originate from the same catchment area as the cases to be studied. Recall bias tends to be similar in both groups, as they have been equally exposed to thought-provoking questioning by health care professionals and are equally likely to have speculated about causes of their disease. Compliance should be similar and follow-up better, because current contact information is available for cancer patients. An alternative comparison group involves the use of population controls who are usually selected from population rolls, such as those maintained by provincial health care agencies. However, addresses and telephone numbers in these data files are out-of-date for a large proportion, who may not have had medical problems requiring consultation for some time. In addition, the use of population controls is very expensive.

To estimate the effects of SES on risk for lung cancer, all other cancers were used as controls. In analyzing effects for other sites, odds ratios (OR), were estimated using two discrete control groups: 1) all sites except the site of interest and 2) all sites except the site of interest and lung cancer.

Lung cancer was excluded because the strength of the association between this cancer and cigarette smoking makes it difficult to adjust for statistically. If errors in reporting smoking have occurred or the stratification levels selected were inappropriate, residual confounding may result. Calculation of risk estimates both with and without including lung cancer cases in the control group allowed comparison of the estimates. Although the direction and magnitude of the risk estimates were similar, those based on the exclusion of lung cancer cases were generally more precise. Where the probability of error varied with type of control, the majority of the probabilities were lower for the group excluding lung cancers. Examples of selected differences in risk estimates and precision are shown in Table 3.1.

Traditionally, when using cancer controls, one or two specific sites are selected, so that the same control group is used for all the cases. This was not possible in this analysis because a significant number of colon, prostatic, testicular and non-melanotic skin cancers, participating in other studies, were excluded (please see Table 2.1). This reduced the pool of possible control cancers to such an extent that it was necessary to include all sites except the index site and lung cancer, even though this meant a different control group for each cancer site.

The reference group for the unadjusted estimates was all education levels excluding the index level (in subsequent OR analyses, the reference group was always the lowest category for that variable to maximize differences). The equations for the multiple logistic regressions in this table included the possible confounders, smoking status and alcohol consumption. All other risk estimates not listed in Table 3.1 were quantitatively similar for both control groups. Therefore, subsequent analyses excluded lung cancer cases from the control group.

Table 3.1

Risk Estimates Including or Excluding Lung Cancer Controls

Education Level	Cancer	Lung Cancer Included Unadjusted OR			Lung Cancer Excluded Unadjusted OR		
		OR	95% CI	p	OR	95% CI	p
Elementary	Malignant Melanoma	0.23	0.16-0.34	0.0000	0.25	0.17-0.36	0.0000
	Oesophageal	2.20	1.15-4.20	0.01	2.33	1.22-4.45	0.008
Secondary	Prostatic	0.84	0.74-0.97	0.14	0.85	0.74-0.98	0.02
	Kidney	0.70	0.51-0.95	0.02	0.71	0.52-0.96	0.03
Technical/Vocational	Prostatic	0.76	0.66-0.88	0.000	0.74	0.64-0.86	0.0000
	Testicular	1.53	1.06-2.22	0.02	1.51	1.04-2.19	0.03
University	Lip	0.25	0.12-0.54	0.000	0.23	0.11-0.50	0.0000
	Malignant Melanoma	2.76	2.16-3.53	0.000	2.56	2.00-3.27	0.0000
		Multiple Logistic Regression			Multiple Logistic Regression		
Elementary	Oesophageal	1.87	0.98-3.59	0.06	1.96	1.02-3.75	0.04
	Prostatic	1.25	1.11-1.42	0.0002	1.34	1.18-1.51	0.0001
Technical/Vocational	Prostatic	0.87	0.71-1.03	0.06	0.85	0.73-0.99	0.03
University	Lip	0.26	0.12-0.56	0.0005	0.24	0.11-0.53	0.0003
	Prostatic	0.86	0.71-1.03	0.10	0.80	0.66-0.96	0.02
	Malignant Melanoma	1.71	1.31-2.23	0.0001	1.65	1.27-2.14	0.0002

B. STATISTICAL ANALYSIS

Data management was done using SAS and the UNIX operating system utilities. SAS allows the computation of single and stratified tables and unconditional logistic unconditional linear regression with multiple variables. To estimate the effects of each SES index on cancer at individual sites, several levels of standard case-control methods were used[94]. Unadjusted ORs were calculated separately for the various levels of education, income and occupational status. As cases and controls were not matched in any way *a priori*, stratified analysis was then applied, with adjustment for age, smoking, alcohol and other variables shown to be of interest by the descriptive analysis. Adjustment for age was expected to control also for effects from different year of first employment. As wages in general have increased over time, men beginning their employment in earlier years would be at a disadvantage in lifetime average annual income, compared to men beginning employment more recently. Age and age at first employment are highly correlated, so control for age should also reduce bias due to first employment date.

Finally, on the basis of the results of the stratified analysis, various unconditional regression models were applied, to allow risk estimation with allowance for confounding by other variables. The advantage of applying both stratified and multivariate analysis is that each method has distinctive strengths and limitations. Stratification allows the investigator to develop a real feeling for the data and to assess what is happening at each level of stratification. It is also easier for readers to understand. On the other hand, stratification cannot deal with large numbers of variables at once, without reducing the frequencies in each cell to low numbers. While multivariate analysis is usually very efficient in dealing with large numbers of values, its primary limitation is that the assumption is made that a given mathematical form describes study variable relations. If this model is incorrect in describing the conditional distribution over a range of values for which observations are missing, unquantifiable bias may result. Many epidemiologists recommend the use of both techniques, which complement each other in providing useful insights[94-96].

The particular model applied for unconditional multiple linear logistic regression with binary variables was the parallel lines regression or proportional odds model, discussed by Cox and Snell[97]. The model requires an individual's response to take one of two possible values, denoted for convenience by 1 and 2 (for example, $Y=1$ if a disease is present; otherwise $Y=2$). Where **x** is a vector of explanatory variables and $p = \Pr(Y=1/\mathbf{x})$ is the response probability to be modelled, the linear logistic model takes the form

$$\mathrm{logit}(p) = \log(p/(1-p)) = \alpha + \beta'\mathbf{x}$$

where α is the intercept parameter and β is the vector of slope parameters. The logistic model shares a common feature with a more general class of linear models, that the response variable is assumed to be linearly related to the explanatory variables. Since the mean μ implicitly depends on the stochastic behaviour of the response, and the explanatory variables are assumed fixed, a general function of the mean, which is the logarithm of the odds radio, provides the link between the stochastic random component and the systematic or deterministic component of the response variable Y[98].

To describe the relationship between the three measures of SES, multi-way cross-tabulations were done using the SAS PROC FREQ procedure[86], which produces frequencies and statistical analysis. Measures of association computed are chi square, probability and several coefficients of correlation.

To assess their relative contributions to the magnitude of risk, multiple linear logistic regression using backward elimination was applied to cancer site-risk factor combinations which were statistically significant after modelling each individual SES index and with age, smoking status and alcohol consumption. In this procedure, all parameters of interest are entered into the model and the least significant variable is removed. The process is repeated until no remaining variables meet the specified level for removal ($p>0.05$).[98]

Effect modification and interaction were assessed using two different methods. Effect modification was assessed using multiplicative models and multiple logistic regression analysis to evaluate independence of effects. In multiplicative models the rate ratio is assumed to be uniform over the strata employed. To evaluate possible interaction between the SES indices, age, smoking and alcohol, the method described by Rothman[96] was applied. A mathematical model such as logistic regression applies arbitrary criteria to evaluate joint effects and may not detect any interaction if the effects are proportional. Rothman's epidemiologic estimation, on the other hand, assumes that for meaningful evaluation of interaction, an appropriate epidemiologic definition of independence must be applied: that is, if two sufficient causes act only through different sufficient causes, their actions are epidemiologically and biologically independent.

Applying this method produces the relative excess risk due to interaction (RERI), a synergy index with confidence limits (S) and a measure of the proportion of disease attributable to interaction (AP*).[96] Where RR represents the rate ratio for disease, A and B are factors, then

$$\text{RERI} = \text{RR(AB)-RR(AB-RR(}\bar{\text{A}}\text{B)+1}$$

and

$$\text{S} = \frac{\text{RR(AB)-1}}{\text{RR(AB) + RR (}\bar{\text{A}}\text{B)-2}}$$

and

$$\text{AP*} = \frac{\text{RR(AB) - RR (AB) -RR (}\bar{\text{A}}\text{B)+1}}{\text{RR (AB) - 1}}$$

Calculation of the confidence limits involved the ratio of the risk among those in the combined exposure category to the risk among those unexposed in both categories[100]. Unadjusted odds ratios were estimated using the Mantel-Haenszel method, while stratified analysis employed Woolf's method for combined ratios. Confidence limits were based on the normal distribution. The methods are described in detail by Breslow and Day[94].

PROC REG[98] was applied to test for trends using Students' *t*. This regression procedure prints the *t* ratio, the significance probability and also the coefficient of determination, R^2. This is the proportion of variation in the dependent variable that is predictable from the independent variable. The homogeneity chi square statistic was not used to test for trend because sparsity of data resulted in too many zero frequencies, when age, smoking and alcohol consumption were included as stratification variables.

CHAPTER IV

RESULTS

A. EDUCATION

Unadjusted ORs were estimated for secondary education, technical/vocational training and university education, compared to the baseline, elementary education, which was assigned a value of 1 in the computations. For sites with 5 cases or more, statistically significant unadjusted ORs were then stratified by age (≤ 60, >60), cigarette smoking status (Ever or Never Smoked) and alcohol consumption (Ever or Never Drank Alcohol). "Never" was defined as smoking or drinking for more than three months consecutively. Multiple logistic regression analysis was applied to education level - cancer site associations which remained statistically significant after stratified analysis. Education level, age, smoking status and alcohol consumption were dichotomized in the analysis; the results are shown in Table 4.1 as described above.

Compared to men with an elementary education only, those with secondary, technical/vocational or university education enjoyed statistically significant deficits in cancers of the lip, stomach, lung and prostate (Table 4.1). Risk estimates for these cancers among those with more than an elementary education were generally half to three quarters as high as the risk estimates among those with elementary education only and the confidence intervals all excluded 1. Men with technical/vocational or university education also had significantly lower risks for laryngeal cancer (ORs = 0.51 and 0.38 respectively, 95% CIs - 0.32 - 0.82 and 0.21 - 0.69 respectively).

Table 4.1

Statistically Significant Risk Estimates[2]
Associated With Educational Attainment

Education Level	Cancer	Cases	Multiple Logistic Regression		
			O.R.	95% CI	P
Secondary	Lip	37	0.48	0.32-0.71	<0.01
	Stomach	45	0.58	0.40-0.84	<0.01
	Lung	215	0.79	0.66-0.95	0.01
	Prostatic	317	0.78	0.66-0.92	<0.01
	Malignant Melanoma	74	2.76	1.75-4.35	<0.01
	Non-Melanoma	310	1.68	1.40-2.03	<0.01
	Testicular	40	3.36	1.61-7.02	<0.01
Technical	Lip	20	0.28	0.17-0.47	<0.01
	Stomach	40	0.58	0.39-0.87	0.01
	Laryngeal	28	0.51	0.32-0.82	0.01
	Lung	167	0.70	0.57-0.86	<0.01
	Prostatic	268	0.75	0.63-0.89	<0.01
	Malignant Melanoma	76	2.72	1.71-4.33	0.01
	Non - Melanoma	313	1.85	1.53-2.23	0.01
University	Lip	7	0.13	0.06-0.29	<0.01
	Stomach	25	0.51	0.32-0.82	0.01
	Laryngeal	14	0.38	0.21-0.69	<0.01
	Lung	56	0.37	0.27-0.49	<0.01
	Prostatic	161	0.65	0.52-0.80	<0.01
	Malignant Melanoma	99	4.71	2.98-7.47	<0.01
	Non-Melanoma	308	2.60	2.13-3.18	<0.01

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[2] Reference for all risk estimates was Elementary Education, OR=1.

Men with an elementary education were at a significant risk advantage for skin cancers, both malignant melanoma and NMSC, with risk at both sites increasing directly with level of education. Men with secondary or technical/vocational education were at approximately twice the risk of NMSC and three times the risk of malignant melanoma, while ORs for university-educated men were 2.60 (95% CI = 2.13 - 3.18) for NMSC and 4.71 (95% CI = 2.98 - 7.47) for malignant melanoma. Men with a secondary education were also at significantly increased risk of testicular cancer (OR = 3.36, 95% CI = 1.61 - 7.02), compared to those with elementary education only.

B. INCOME

Average annual earned, imputed income was divided into three categories: <\$10,000 per year over an employment lifetime, \$10,000 to \$19,999 and ≥\$20,000. With the standard income against which other levels were compared set at <\$10,000, unadjusted and stratified ORs were estimated for the other income categories. The ORs which remained statistically significant after control for age, smoking status and alcohol consumption were modelled as described for education level; the results appear in Table 4.2. Men in the upper two income categories had statistically significant deficits of lip and prostatic cancers. Men in the highest income category also had an advantage in risk for lung cancer (OR = 0.56, 95% CI = 0.43=0.73), compared to those in the lowest category. The upper income categories had excess risks for NMSC and malignant melanoma, following the pattern of excess risks in upper educational levels, compared to the standard. Men earning <\$20,000 per year also were at excess risk for testicular

(OR = 8.04, 95% CI = 2.89 - 22.36) and brain cancers (OR = 2.07, 95% CI = 1.13 - 3.81) and for Hodgkin's disease (OR = 4.56, 95% CI = 2.21 - 9.40), compared to men earning <$10,000 per year. Earned income was also analyzed weighted by the cost-of-living index for each year. The resulting ORs were of the same approximate

Table 4.2
Statistically Significant Risks Associated With Mean Annual Lifetime Income[3]

Income Level $	Cancer	Cases	Multiple Logistic Regression		
			Odds Ratio	95% Confidence Interval	P
10,000-19,999	Lip	50	0.42	0.28-0.62	<0.01
	Prostatic	428	0.68	0.59-0.79	<0.01
	Malignant Melanoma	117	1.95	1.32-2.88	<0.01
	Non-Melanoma	516	1.49	1.28-1.74	<0.01
≥20,000	Lip	11	0.15	0.08-0.30	<0.01
	Lung	111	0.56	0.43-0.73	<0.01
	Prostatic	171	0.59	0.48-0.72	<0.01
	Malignant Melanoma	141	2.72	1.80-4.12	<0.01
	Non-Melanoma	370	1.53	1.26-1.85	<0.01
	Testicular	90	8.04	2.89-22.36	<0.01
	Brain	61	2.07	1.13-3.81	<0.01
	Hodgkin's Disease	66	4.56	2.21-9.40	<0.01

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[3] Odds ratios were estimated using as a reference group the lowest income category, <$10,000, OR=1.

magnitude in the same direction and of similar statistical significance both before and after weighting. The exceptions were cancers of the liver, gallbladder, bone and connective tissues, thyroid gland, and Hodgkin's disease, non-Hodgkin's lymphoma and leukaemia. As the number of cases at these sites was small, the differences did not follow a consistent pattern and the ORs were not statistically significant, the cost-of-living weighted analysis was not included in this report.

C. OCCUPATIONAL PRESTIGE RANK

The occupational prestige rank associated with each individual's final employment was selected for analysis, rather than a composite index of ranks over a lifetime.

Based on the lowest prestige rank[16] as the reference category, ORs were also estimated for the 15 remaining Pineo-Porter occupational prestige ranks. Multiple logistic regression was applied to those prestige rank - cancer site combinations for which the associations remained statistically significant after stratification for age, then smoking status, then alcohol consumption. These variables were also included in the model. The results are shown in Table 4.3.

After control for age, smoking and alcohol consumption, risk for prostatic cancer in the other prestige ranks was generally about 50% to 60% of the risk in farm labourers (Table 4.3). The exceptions were the categories of unskilled clerical/sales/service (rank 14, OR = 0.64, 95% CI = 0.40 - 1.01, n = 27) and/or farm owner/operator (rank 11, OR - 0.83, 95% CI = 0.66 - 1.06, n = 252). ORs for these prestige ranks were also below unity, but not significantly so. Farm labourers also had high rates of lip cancer, compared to semi-skilled manual labourers (OR = 0.43, 95% CI = 0.21 - 0.86), workers in skilled crafts/trades (OR = 0.30, 95% CI = 0.15 - 0.57) and employed professionals (OR = 0.23, 95% CI = 0.10 - 0.54). The deficits were well below unity, regardless of differences in ranking (10 & 13 versus 2 respectively).

There were also differences in risk for stomach cancer which did not follow a consistent pattern according to rank. Semi-skilled clerical/sales/service workers, ranked 12, and farm owners/operators, ranked 11, had significant deficits in risk (ORs = 0.32 and 0.39, 95% CIs = 0.13 - 0.79 and 0.21 - 0.71 respectively), as did employed professionals, ranked 2 (OR = 0.32, 95% CI = 0.17-0.70). Stomach cancer ORs for other prestige ranks were all below unity, compared to farm labourers, but not significantly so, nor was there a discernable pattern of risk by rank order. Because the prestige rank scale was relatively large, data were missing in over half the cells and ORs could not be calculated.

Employed professionals had a pronounced deficit of lung cancer, compared to farm labourers (OR = 0.55, 95% CI = 0.35-0.86), even when the effects of smoking, age and alcohol were controlled. Otherwise occupational prestige rank had no effect on lung cancer risk.

Table 4.3
Statistically Significant Risks[4] Associated With Occupational Prestige Rank

Occupational Prestige		Cancer	Cases	Multiple Logistic Regression		
Rank	Category			O.R.	95% CI	P
15	Unskilled Labourer	Prostatic	124	0.62	0.46-0.83	<0.01
		Non-Melanoma	84	3.41	2.08-5.58	<0.01
14	Unskilled Clerical/Sales/Service	Non-Melanoma	23	5.45	2.90-10.25	<0.01
13	Semi-Skilled Manual Labour	Lip	13	0.43	0.21-0.86	<0.02
		Prostatic	110	0.53	0.40-0.72	<0.01
		Nasopharynx/Nasal Sinuses	14	6.43	1.44-28.83	<0.02
		Non-Melanoma	99	4.31	2.64-7.03	<0.01
12	Semi-Skilled Clerical/Sales/Service	Stomach	6	0.32	0.13-0.79	<0.01
		Prostatic	64	0.58	0.41-0.83	<0.01
		Non-Melanoma	57	4.54	2.69-7.64	<0.01
11	Farm Owner/Operator	Stomach	19	0.39	0.21-0.71	<0.01
		Non-Melanoma	199	6.29	3.95-10.01	<0.01
10	Skilled Crafts/Trades	Prostatic	177	0.52	0.40-0.68	<0.01
		Lip	15	0.30	0.15-0.57	<0.01
		Non-Melanoma	199	5.11	3.21-8.12	<0.01

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[4] Odds ratios were estimated using as a reference group the category ranked 16 - Farm Labourer, OR=1.

Table 4.3 (cont'd)

Statistically Significant Risks Associated With Occupational Prestige Rank

Occupational Prestige		Cancer	Cases	Multiple Logistic Regression		
Rank	Category			O.R.	95% CI	P
9	Skilled Clerical/Sales/Service	Prostatic	38	0.52	0.34-0.79	<0.01
		Non-Melanoma	57	7.42	4.37-12.60	<0.01
4	Semi Professional	Prostatic	24	0.50	0.30-0.84	0.01
		Malignant Melanoma	16	2.87	1.26-6.55	0.01
		Non-Melanoma	46	8.42	4.85-14.64	0.01
3	Upper Manager	Prostatic	84	0.60	0.44-0.83	<0.01
		Non-Melanoma	104	6.75	4.14-11.01	<0.01
2	Employed Professional	Lip	7	0.23	0.10-0.54	<0.01
		Stomach	12	0.35	0.17-0.70	<0.01
		Lung	35	0.55	0.35-0.86	0.01
		Prostatic	107	0.63	0.47-0.86	<0.01
		Non-Melanoma	159	7.56	4.71-12.12	0.01
		Malignant Melanoma	48	2.53	1.28-4.99	0.01
1	Self-Employed Professional	Prostatic	18	0.46	0.26-0.80	0.01
		Non-Melanoma	34	9.23	5.10-16.68	<0.01

On the other hand, farm labourers enjoyed a favourable experience with skin cancer. ORs for every other rank were statistically significantly high in relation to farm labourers. ORs ranged from about three and one-half to over nine times the ORs among farm labourers. Only two ranks had excess risks for cutaneous malignant melanoma compared to farm labourers: semi-professionals, ranked 4 (OR = 2.87, 95% CI = 1.26-6.55) and employed professionals, ranked 2 (OR = 2.53, 95% CI = 1.28-4.99).

Finally, there were some excesses among middle-ranked prestige levels. Semi-skilled manual labourers showed an excess of cancer of the nasopharynx or nasal sinuses (OR = 6.43, 95% CI = 1.44-28.83), middle managers an excess for brain cancer (OR = 3.59, 95% CI = 1.17-11.01), and semi-skilled manual workers an excess of laryngeal cancer (OR = 2.49, 95% CI = 1.12-5.56). These excesses were limited to one prestige rank each, without a discernible pattern.

D. RELATIONSHIPS BETWEEN VARIABLES

The SAS PROC FREQ procedure lists phi and contingency coefficients of correlation, as well as Cramér's V.[101] The latter is shown in Table 4.4 because it more accurately reflects the strength of an association where the cross tabulation is larger than two by two. Cramér's V ranges always from -1 to +1, whereas phi and the contingency coefficients are dependent on sample size and may exceed 1. This may be misleading, suggesting a stronger association between variables than is actually the case.

As Table 4.4 shows, education, imputed earned income and occupational prestige were positively associated, as were cigarette smoking and alcohol consumption. All the correlations were statistically significant. However, although the associations between the SES variables were significantly different from zero, the relatively small size of the correlation does not guarantee the high degree of relationship necessary for efficient predictive ability between variables. Using Guilford's suggested scale of coefficient magnitude to interpret degree of relationship, the association between education and income was low, while the association between both education and income and occupational prestige was more substantial.[102] Against expectations, the correlations between smoking or alcohol and the SES indices were not high. With a few exceptions, available literature shows the number of non-smokers and ex-smokers rises with both income and education level and the proportion of current smokers is higher at lower educational levels.[14,48,49,74,75] The exceptions are recent findings that current smokers are more likely to be employed and at a higher socioeconomic level[103,104] and that low income and low smoking rates are closely associated among United States blacks.[105] Published results on alcohol consumption are not readily available. Although Canada's Health Promotion Survey found high income people were more likely to drink heavily[14], a recent Italian study reported wine consumption was evenly distributed throughout the various education levels.[106] The expectation would be that at least smoking should correlate well with income and education in the Alberta data.

E. COLLAPSED OCCUPATIONAL PRESTIGE SCALE

At sixteen levels, the occupational prestige variable was refined and numbers at each level were reduced dramatically. Therefore, the index was collapsed into 6 levels, as suggested by Pineo, Porter and McRoberts.[80] The 16-level and 6-level indices correlated perfectly, as shown in Table 4.4, so multiple linear logistic regression was applied to the 6-level prestige index. The results of the regression, with blue collar labourers as the baseline and controlling for age, smoking and alcohol consumption, appear in Table 4.5. Lip cancer was significantly low, compared to blue collar workers, in managers/professionals, supervisors/middle management, and lower white collar workers, with ORs of 0.29 (95% CI = 0.15-0.57), 0.40 (95% CI = 0.20-0.79), and 0.45 (95% CI = 0.28-0.74). Unexpectedly, farm owners/operators were at increased risk of lip cancer (OR = 1.81, 95% CI 1.18-2.76), compared to blue collar workers. There was a significant deficit of stomach cancer among managers/professionals (OR = 0.53, 95% CI 0.33-0.84) and of laryngeal cancer among farm owners (OR = 0.45, 95% CI = 0.23-0.87). Significant deficits in lung cancer were found among upper managers/professionals (OR = 0.47, 95% CI = 0.36-0.62) and supervisors/middle management (OR = 0.71, 95% CI = 0.55-0.92), while the latter also had a lower risk for non-Hodgkin's lymphoma.

Non-melanotic skin cancer was 1.8 to 2.5 times higher in all collapsed prestige groups, compared to farm labourers. Significant excesses of malignant melanoma were also found in the upper four collapsed prestige categories ranks IV-V inclusive. Excess risks increased directly with occupational prestige, from an OR of 1.59 in lower white collar workers to an OR of 2.16 in the managerial/professional group.

Table 4.4

Strength of Association[5] Between SES Variables

	Education	Income	Occupational Prestige	Collapsed Occupational Prestige	Age	Smoking	Alcohol
Education	1.00	0.34	0.40	0.36	0.17	0.13	0.05
Income		1.00	0.47	0.42	0.41	0.11	0.09
Occupational Prestige			1.00	1.00	0.17	0.13	0.05
Collapsed Occupational Prestige				1.00	0.12	0.10	0.11
Age					1.00	0.18	0.06
Smoking						1.00	0.33
Alcohol							1.00

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[5] Coefficient of correlation = Cramér's V.

Table 4.5
Statistically Significant Risks Associated With Collapsed Occupational Prestige Scale

Prestige Level	Cancer	Cases	Multiple Logistic Regression		
			Odds Ratio[6]	95% C.I.	P
I. Managerial/Professional	Lip	10	0.29	0.15-0.57	<0.01
	Stomach	26	0.53	0.33-0.84	<0.01
	Lung	75	0.47	0.36-0.62	<0.01
	Malignant Melanoma	82	2.16	1.48-3.13	<0.01
	Non-Melanoma	297	2.46	2.02-3.00	<0.01
II. Semi-Professional/Technician	Malignant Melanoma	25	2.31	1.35-3.93	<0.01
	Non-Melanoma	68	2.40	1.74-3.31	<0.01
III. Supervisory/Middle Management	Lip	10	0.40	0.20-0.79	0.01
	Lung	86	0.71	0.55-0.92	0.01
	Non-Hodgkin's Lymphoma	18	0.53	0.31-0.90	0.02
	Malignant Melanoma	41	1.63	1.06-2.52	0.03
	Non-Melanoma	166	1.88	1.50-2.36	<0.01
IV. Lower White Collar	Lip	25	0.45	0.28-0.74	<0.01
	Prostatic	306	0.78	0.66-0.93	0.01
	Malignant Melanoma	83	1.59	1.11-2.29	0.01
	Non-Melanoma	336	1.82	1.51-2.20	<0.01
V. Farm Owners	Laryngeal	13	0.45	0.23-0.87	0.02
	Lip	46	1.81	1.18-2.76	0.01
	Non-Melanoma	199	2.08	1.67-2.60	<0.01

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[6] Odds ratios were estimated using as a reference group the category ranked VI - Blue Collar Labourer, OR=1.

F. TESTS FOR TREND AND EXPLANATION OF VARIATION IN RISK

To assess exposure-response trends, *t* tests for trends in the ORs were applied, controlled for age, smoking and alcohol. The amount of response variation explained by each model was also estimated by the coefficient of determination.

In univariate analysis, education was significantly associated with risk for cancer at several sites. Risk decreased as educational attainment increased for cancers of the lip, stomach, lung, prostate and larynx. Direct increases in risk with increasing education were seen for malignant melanoma, NMSC and testicular cancer. However, when the ORs were adjusted for age, smoking and alcohol, there were no statistically significant trends in risk by education level for any site of cancer (Figures 1-8). At the same time, a large part of the variation in risk for disease was explained by education: over 80% of the variance in risk for cancers of the lip, lung, larynx, testis and NMSC, and over 70% for prostate cancer and malignant melanoma. Only for stomach cancer was relatively little of the variation in risk explained by education (Figures 1-8).

Earned, imputed income and disease exhibited statistically significant trends for some cancer sites, but not for all the sites which were significantly associated with income in univariate analysis (Figures 9-16). Trends for risk to decrease as income rose were significant for cancers of the prostate and lung; a significant trend was also found between risk for brain cancer and increasing income. A high proportion of the variation in risk by income was explained for all these sites, except NMSC (Figure 12).

Analysis of trends was repeated for those cancer sites where risk was significantly associated with collapsed prestige rank in univariate analysis (Figures 17-22). Only risk for laryngeal cancer increased significantly as occupational prestige decreased; 89% of the variance in risk was explained by prestige in this analysis. Trends in risk for the other cancers were not statistically significant, although the collapsed prestige scale explained 70% and 65% of the variation in risk for lip cancer and NMSC respectively and the p-values were close to being significant ($p=0.08$ and 0.10 respectively).

Tests for Trend & Variance Explained

Figure 1. Lip Cancer

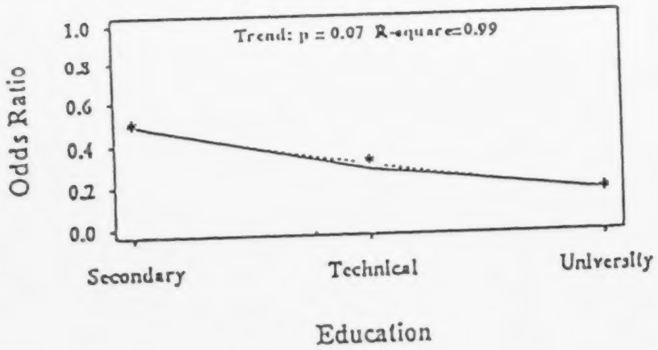


Figure 2. Stomach Cancer

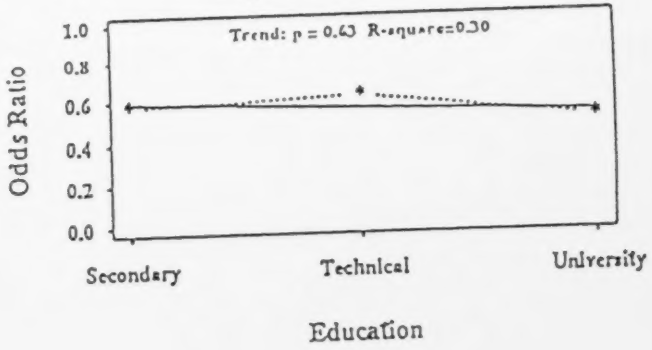


Figure 3. Lung Cancer

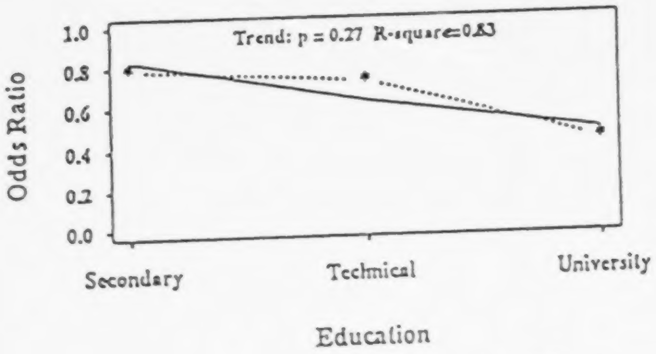
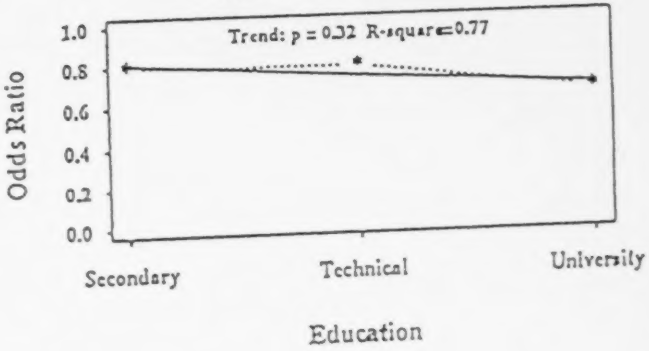


Figure 4. Prostatic Cancer



Legend ...... Adjusted Odds Ratios ___ Trend

Figure 5. Laryngeal Cancer

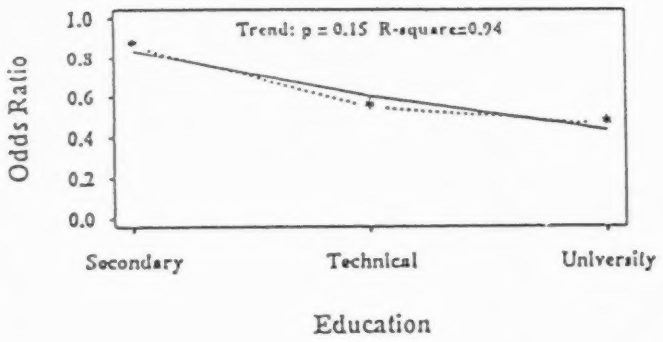


Figure 6. Melanotic Skin Cancer

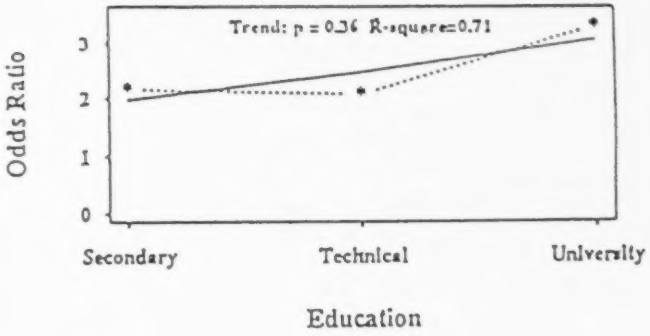


Figure 7. Non-Melanotic Skin Cancer

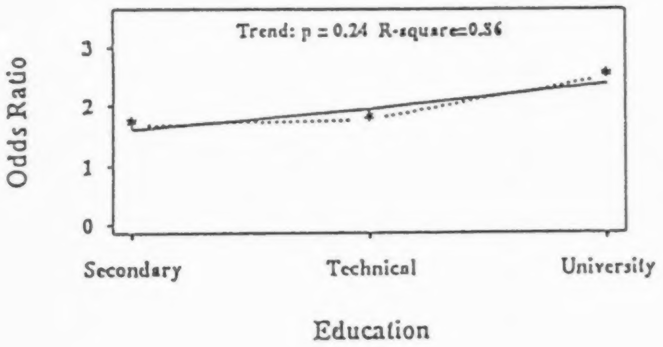
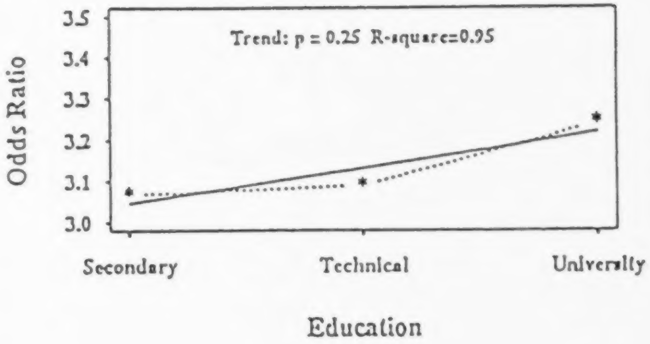


Figure 8. Testicular Cancer



Legend ...... Adjusted Odds Ratios ____ Trend

Figure 9. Lip Cancer

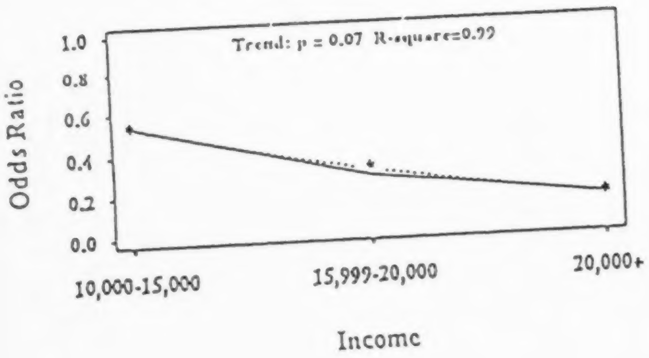


Figure 10. Prostatic Cancer

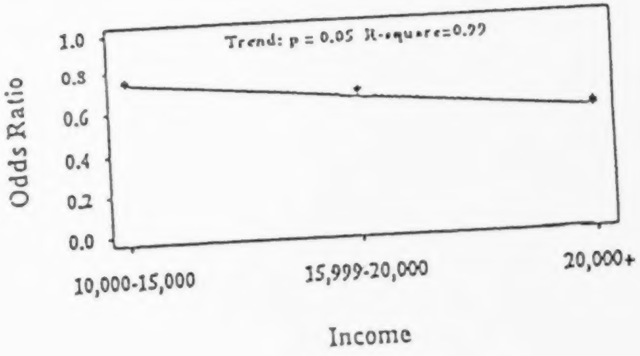


Figure 11. Lung Cancer

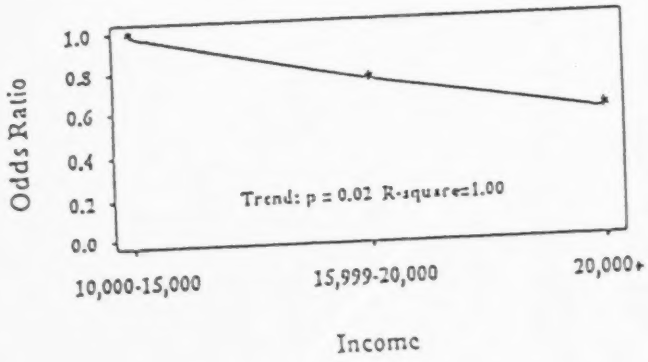
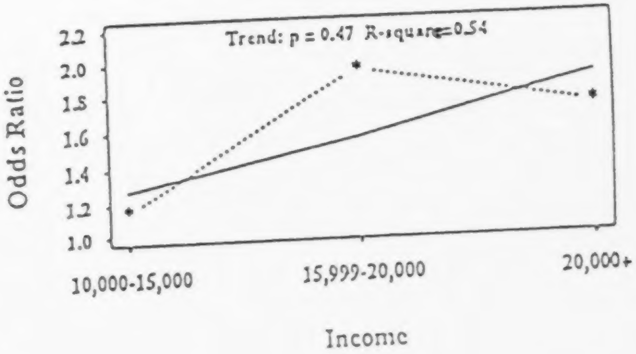


Figure 12. Non-Melanotic Skin Cancer



Legend ...... Adjusted Odds Ratios ___ Trend

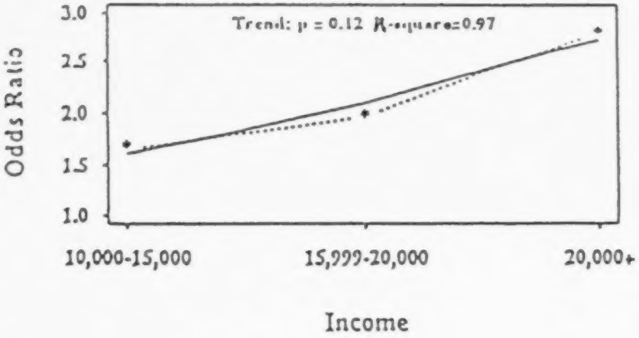


Figure 13. Melanotic Skin Cancer

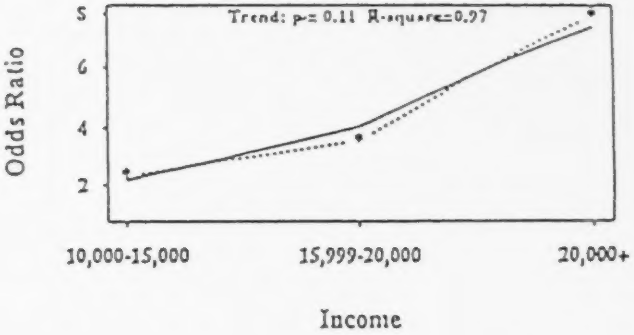


Figure 14. Testicular Cancer

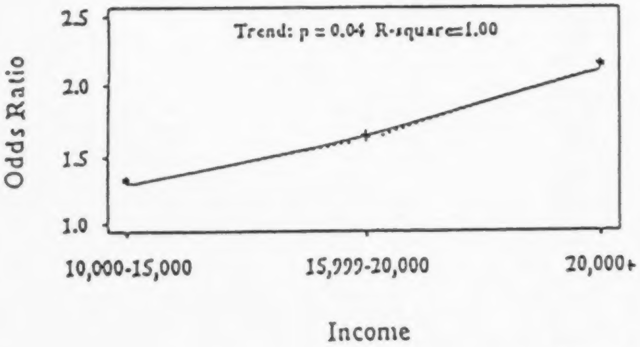


Figure 15. Brain & CNS Cancer

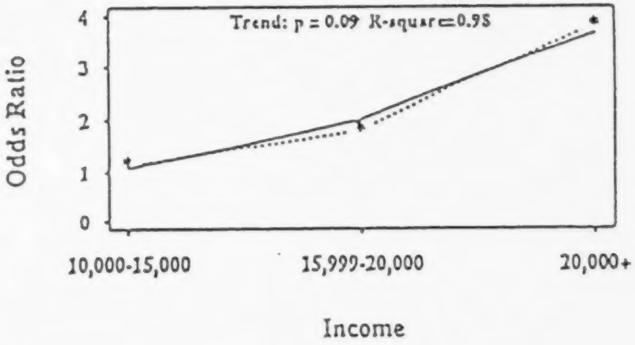


Figure 16. Hodgkin's Disease

Legend ...... Adjusted Odds Ratios ____ Trend

Figure 17. Lip Cancer

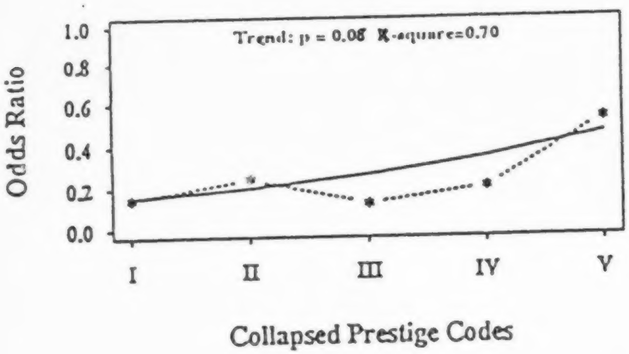


Figure 18. Stomach Cancer

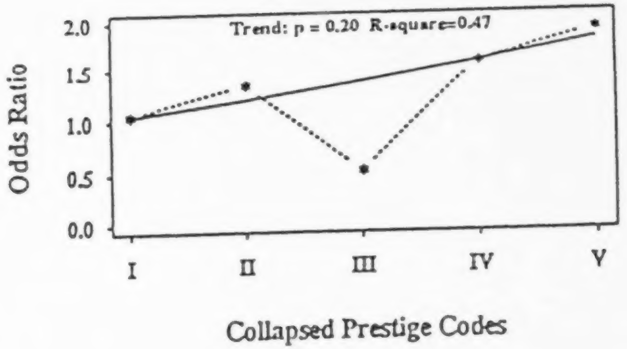


Figure 19. Laryngeal Cancer

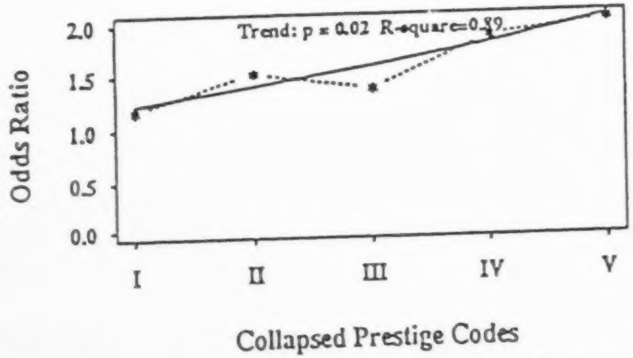
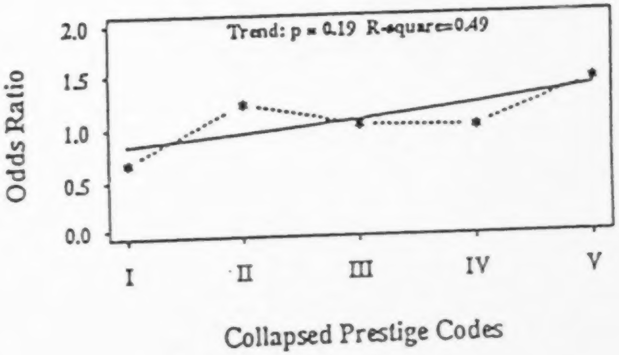


Figure 20. Lung Cancer



Legend ...... Adjusted Odds Ratios ___ Trend

Figure 21. Non-Hodgkin's Lymphoma

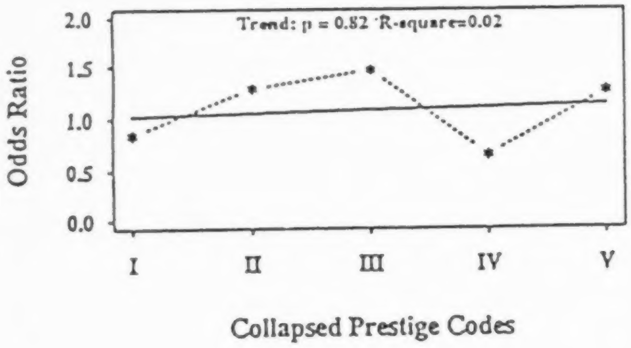


Figure 22. Non-Melanotic Skin Cancer

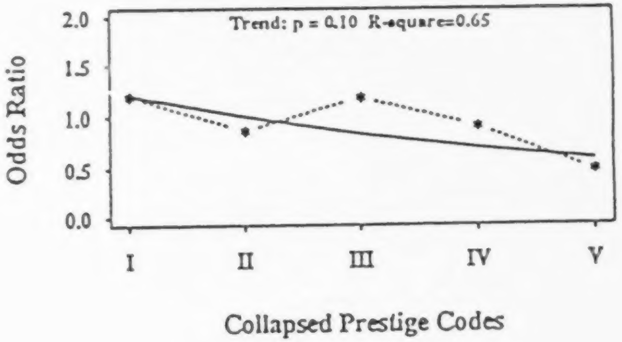
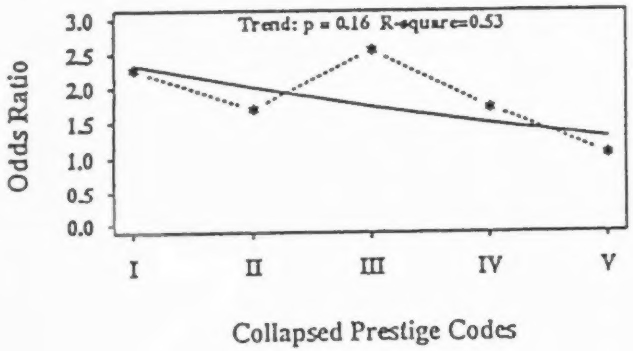


Figure 23. Melanotic Skin Cancer



Legend ...... Adjusted Odds Ratios ____ Trend

G. RELATIVE CONTRIBUTIONS TO RISK ESTIMATES

Statistical significance in goodness-of-fit tests varies with sample size. To facilitate substantive assessment, estimation of model parameters and descriptions of associations should use a variety of methods for better understanding of the data. The coefficient of determination is also highly dependent on the distribution and range of the component variables. It is interpretable when the sample is large and the relationship between the variables is approximately linear[96].

To clarify the relative contribution to cancer risk of the SES indices at each site and of age, cigarette smoking and alcohol, multiple linear logistic regression with backward elimination was applied to cancer site-variable combinations which remained statistically significant after modelling with the individual SES indices, previously described in sections A, B and E (the model is specified in Appendix V). For each cancer site, age was dichotomized close to the median of the age distribution for that site. Interaction was assessed using Rothman's epidemiologic estimation method.[96]

Lip Cancer

For lip cancer, age (≤ 50, >50), income ($<10{,}000$, 10,000-19,999, $\geq 20{,}000$), education (elementary, secondary/technical, university) and collapsed occupational prestige scale (ranks VI-V, IV-I inclusive) were inserted in the equation (Table 4.6). For each independent variable comparison, baseline was the lowest level or rank. The ORs associated with education decreased as education level increased, from 0.48 for secondary/technical education (95% C.I. = 0.35-0.70, $p = 0.0001$), to 0.25 for university (95% C.I. = 0.12-0.54, p = 0.0001). However, the overall trend of risk associated with education was not statistically significant.

An occupation in the Managerial/Professional or Lower White Collar groups (prestige ranks I-IV) reduced risk for lip cancer to about half or less that among blue collar workers. Otherwise a clear relationship between magnitude of risk and occupational prestige rank was not found. Compared to rank VI, risk for lip cancer decreased about 40% in ranks III and IV, and about 30% in ranks I and II, then increased by 80% in rank V (Table 4.5). When ORs were estimated for lip cancer and the refined occupational prestige ranking, simultaneously stratified by age and cigarette smoking, absence of pattern was even more obvious. Compared to rank 16 (farm labourers), there were significant deficits in risk associated with ranks 2,3 and 6 (ORs = 0.41, 0.33, 0.33; 95% CIs = 0.17-0.98, 0.11-0.98, 0.11-0.98 respectively) and significant excesses associated with ranks 11 and 12 (ORs = 3.01 and 2.51, 95% CIs = 2.10-4.30 and 1.58-3.98). Risk estimates for the other ranks were not significant and bore no relation to rank order.

Table 4.6
Statistically Significant Risk Factors for Cancer

Cancer	Risk Factor	Level of Factor	Multiple Logistic Regression			
			β	p	O.R.	95% C.I.
Lip	Education	Secondary/Technical	0.73	0.0001	0.48	0.33-0.70
		University	1.37	0.0004	0.25	0.12-0.54
	Occupational Prestige	Ranks IV-I	0.67	0.0006	0.51	0.35-0.75
	Age	≥ 50	0.72	0.0012	0.49	0.31-0.75
	Income	$\geq 20,000$	0.96	0.0059	0.38	0.19-0.76
Head & Neck Excluding Lip	Age	> 55	0.52	0.0010	0.59	0.44-0.81
	Smoking	Yes	-0.54	0.0189	1.72	1.09-2.71
Stomach	Education	Secondary/Technical	0.64	0.0001	0.52	0.38-0.72
		University	0.74	0.0005	0.48	0.31-0.72
	Smoking	Yes	-0.65	0.0069	1.91	1.19-3.06
	Occupational Prestige	Rank V	0.58	0.0209	0.56	0.34-0.92
Laryngeal	Smoking	Yes	-2.75	0.0001	15.69	3.88-63.52
	Education	University	0.73	0.0041	0.48	0.30-0.79
		Secondary/Technical	0.57	0.0145	0.57	0.36-0.89
	Occupational Prestige	Rank V	0.67	0.0281	0.51	0.28-0.93

Table 4.6 (cont'd)

Statistically Significant Risk Factors for Cancer

Cancer	Risk Factor	Level of Factor	Multiple Logistic Regression			
			β	p	OR	95% CI
Lung	Smoking	Heavy	-1.71	0.0001	5.51	4.25-7.13
		Light	-0.63	0.0001	1.87	1.41-2.48
	Education	University	0.66	0.0001	0.52	0.39-0.68
		Secondary/Technical	0.27	0.0017	0.76	0.65-0.90
	Income	≥20,000	0.28	0.0147	0.75	0.60-0.95
Malignant Melanoma	Age	≥50	1.33	0.0001	0.27	0.20-0.35
	Education	University	-0.87	0.0001	2.39	1.56-3.66
		Secondary/Technical	-0.59	0.0032	1.80	1.22-2.65
	Income	≥20,000	-0.56	0.0063	1.75	1.17-2.61
		10,000-19,999	-0.49	0.0075	1.64	1.14-2.36

Table 4.6 (cont'd)

Statistically Significant Risk Factors for Cancer

Cancer	Risk Factor	Level of Factor	Multiple Logistic Regression			
			β	p	OR	95% CI
Non-Melanoma	Education	University	-0.82	0.0001	2.28	1.87-2.77
		Secondary/Technical	-0.48	0.0001	1.62	1.37-1.91
	Occupational Prestige	Ranks II-I	0.44	0.0001	0.64	0.52-0.79
		Ranks V-III	0.66	0.0001	0.51	0.42-0.63
	Income	≥10,000	-2.28	0.0005	1.33	1.13-1.55
	Age	>55	-0.18	0.0129	1.20	1.04-1.39
Prostatic	Age	>70	-0.68	0.0001	1.97	1.69-2.29
	Income	≥10,000	0.66	0.0001	0.52	0.45-0.59
	Education	University	0.38	0.0001	0.68	0.56-0.83
		Secondary/Technical	0.23	0.0012	0.79	0.69-0.91

Table 4.6 (cont'd)
Statistically Significant Risk Factors for Cancer

Cancer	Risk Factor	Level of Factor	Multiple Logistic Regression			
			β	p	OR	95% CI
Testicular	Age	>30	1.91	0.0001	0.15	0.09-0.24
	Income	≥20,000	-2.49	0.0001	12.03	5.67-25.49
		10,000-19,999	-1.32	0.0006	3.75	1.76-7.96
	Education	Secondary/Technical	-1.12	0.0023	3.08	1.49-6.34
	Occupational Prestige	Ranks IV-I	0.58	0.0069	0.56	0.37-0.85
	Education	University	-1.01	0.0108	2.75	1.26-6.00
Bladder	Smoking	Yes	-0.74	0.0001	2.10	1.54-2.86
	Income	≥20,000	0.32	0.0090	0.73	0.57-0.92
Brain	Age	≥50	1.64	0.0001	0.19	0.13-0.29
	Income	≥20,000	-0.67	0.0217	1.95	1.10-3.45
		10,000-19,999	-0.57	0.0378	1.77	1.03-3.03
		49-64	0.43	0.0189	0.65	0.45-0.93
	Income	≥20,000	-0.55	0.0230	1.73	1.08-2.79
Thyroid	Age	>49	1.65	0.0001	0.19	0.09-0.41

Being older than 50 (OR = 0.49, 95% CI = 0.31-0.75, p = 0.0012) and having a mean yearly lifetime earned income over $20,000.00 (OR = 0.38, 95% CI = 0.19-0.76, p = 0.0059) also contributed to a deficit in lip cancer risk. Income exhibited an exposure-response trend, with risk for lip cancer increasing as income decreased. Compared to an unadjusted OR of 1 among men earning less than $10,000 per year, the OR associated with a mean annual income of $10,000-19,999 was 0.58 (95% CI = 0.40-0.82), while for $20,000 or more, the OR was 0.19 (0.10-0.37). However, the trend was not statistically significant. The association between lip cancer risk and age was not as clear. Although men over 50 enjoyed a deficit in risk in the logistic regression model, which included only two age categories, risk by more restricted age groups did not follow a pattern and all ORs were non-significant.

Estimation of interaction between the variables associated with lip cancer is shown in Table 4.7. None of the synergy indices were statistically significant; the confidence intervals included 1 or the numbers were too small to permit calculation. Accordingly, the relative excess risk attributable to the interaction was less than zero and the attributable proportion could not be meaningfully calculated.

As education, occupational prestige and income are surrogates rather than causal per se, other factors were analyzed in more detail, where data on these were available. The association between lip cancer and smoking was investigated, because other reports have demonstrated both a significant association[107] and equally absence of any association.[108-110]

Table 4.7
Estimation of Interaction Between Risk Factors

Cancer	Variables	Synergy Index	95% Confidence Interval
Lip	Education, Occupational Prestige Rank	0.01	- -
	Education, Age	-2.33	- -
	Education, Income	-0.07	- -
	Age, Income	1.28	0.47-3.46
Head & Neck Except Lip	Age, Smoking	1.19	0.44-3.22
Stomach	Education, Occupational Prestige Rank	1.88	0.23-15.27
Laryngeal	Education, Smoking	0.71	0.47-1.08
Lung	Smoking, Education	1.61	1.32-1.96
	Smoking, Income	1.75	1.16-2.63
	Education, Income	0.77	0.29-2.08
Malignant Melanoma	Age, Education	4.26	1.40-12.98
	Age, Income	2.62	0.49-13.99
	Education, Income	2.42	0.70-8.37
Non-Melanoma	Education, Occupational Prestige Rank	1.14	0.74-1.76
	Education, Income	1.46	0.63-3.38
	Age, Education	0.87	0.65-1.18
	Age, Income	0.66	0.42-1.05

Table 4.7 (Cont'd)
Estimation of Interaction Between Risk Factors

Cancer	**Variables**	**Synergy Index**	**95% Confidence Interval**
Prostatic	Age, Income	1.45	0.92-2.27
	Age, Education	1.01	0.76-1.34
	Income, Education	1.05	0.72-1.53
Testicular	Age, Education	0.81	0.09-7.42
	Income, Education	1.58	0.44-5.75
	Age, Occupational Prestige Rank	1.99	0.78-5.07
Bladder	Smoking, Income	1.41	0.49-4.06
Brain	Age, Income	0.58	0.17-1.94
Hodgkin's Disease	Age, Income	0.59	0.22-1.57

In the Alberta data, smoking was not a statistically significant risk factor after modelling, although unadjusted ORs were significantly high among smokers, compared to men who had never smoked (Table 4.8). More controls than lip cancer cases never smoked (21% versus 12% respectively) and somewhat more cases were heavy smokers (182,626 cigarettes per lifetime), compared to controls (41% versus 37% respectively). More cases were also classed as light (127,837.5 cigarettes per lifetime) or medium smokers (127, 837.6 - 186,277.5 cigarettes per lifetime), compared to controls. Mean number of cigarettes smoked over a lifetime was 2% higher for controls (225,419 cigarettes compared to 221,955 cigarettes among cases), but the difference was not statistically significant (p = 0.30).

Table 4.8

Lip Cancer Risk and Cigarette Smoking

	Number of Cases	Odds Ratio	95% Confidence Interval
Never	17	1	
Light	43	1.79	1.02-3.16
Medium	22	2.04	1.08-3.86
Heavy	57	1.91	1.11-3.30

Smoking habits were compared between levels for each of the SES variables significantly associated with risk for lip cancer in the multiple logistic regression model. Proportions of non-smokers and heavy smokers did not vary markedly between levels for education, occupational prestige, income or age. Mean number of cigarettes smoked varied as would be expected if the indices are surrogates for smoking habits; that is, amount smoked increased with decreases in education, occupational prestige and income, and in general decreased as age increased (Table 4.9).

Workplace exposure may also be a causal or promoting factor for which the SES indices are surrogates. Examination of the occupations subsumed in several of the occupational prestige categories suggests this may be the case. For example, workplace exposures of employed professionals (rank 2), high-level management (rank 3) and middle management (rank 6) are different in both type and degree of hazardousness, compared to the workplace exposures of farm owners/operators (rank 11) and farm labourers (rank 16). The latter two groups are more often exposed to ultraviolet radiation, which may increase risk for lip cancer.[111]

Exposure to sunlight was one of the questions asked in this study. Although few people reported exposure and the results were not statistically significant, ORs for lip cancer associated with sunlight exposure among occupational prestige ranks IV, V and VI (lower white collar, farm owner/operator, blue collar labour), were 0.56, 0.53 and 1.96, respectively. The ORs associated with sunlight exposure among unskilled labourers and farm labourers separately were 2.93 and 5.57 respectively. Men in other occupations did not report any exposure. Although these data are sparse, they are suggestive of an association with sunlight exposure, rather than with occupational prestige.

Table 4.9
Smoking Habits Among Lip Cancer Patients

	Never Smoked	Light Smokers	Medium Smokers	Heavy Smokers	Mean Cigarettes Smoked
Income	N (%)	N (%)	N (%)	N (%)	
< 10,000	9(12)	21(28)	15(20)	30(40)	243,827
10,000-19,999	4(9)	16(36)	4(9)	20(45)	198,212
≥20,000	2(20)	3(30)	2(20)	3(30)	196,778
Education					
Elementary	11(14)	16(21)	14(18)	35(46)	263,834
Secondary	1(3)	19(51)	3(8)	14(38)	180,606
Technical	4(22)	4(22)	4(22)	6(33)	174,720
University	1(17)	2(33)	1(17)	2(33)	180,434
Age					
Age ≤40	1(11)	7(78)	1(11)	-	106,836
41-55	3(9)	12(38)	4(13)	13(41)	194,640
56-65	4(8)	12(25)	10(21)	22(46)	202,199
>65	9(18)	12(24)	7(14)	22(44)	284,940
Occupational Prestige					
Ranks I-IV	5(12)	14(34)	5(12)	17(41)	210,319
Ranks V-VI	10(11)	24(27)	17(19)	37(42)	250,354

There were no clear patterns in relations between self-reported sunlight exposure and risk for lip cancer for either education or income. ORs, again not statistically significant, were: elementary education = 1.08, secondary = 1.43, technical/trade = 1.08, income <\$10,000 = 1.53, \$10,000-19,999 = 0.59, ≥\$20,000 = 0.86.

Cancers of the Head and Neck, Excluding Lip

Age and smoking contributed about equally to risk for cancers of the head and neck other than lip, but in opposite directions; there was no appreciable interaction between the two variables (Table 4.7). None of the SES indices exerted a significant effect. Age ≥55 years was associated with a deficit in risk in the logistic regression model (OR = 0.59, 95% CI = 0.44-0.81, Table 4.6). This appears to be a statistical artifact, as oral and pharyngeal cancers have been previously shown to increase with age.[112] The reason for the reduced risk in the logistic regression model appears to be that the highest risk in this group was among men 46 to 55 years of age. In addition, the frequency of head and neck cancer cases was heavily concentrated between the ages 45 and 55, with 42% of all cases in this age group, compared to 27% of the control group. The net effect was to produce a much reduced risk estimate among men over 55 years of age (Table 4.6). When risk was estimated according to more detailed age groups, compared to men under 30, risk was four-fold higher in men 46 to 55 (Table 4.10), decreasing thereafter to 1.65 in men ≥66. These risk estimates included unity in the confidence intervals.

Table 4.10

Variation In Risk for Head and Neck Cancer By Age

		Unadjusted	
Age	Number of Cases	Odds Ratio	95% Confidence Interval
31-45[7]	24	2.55	0.60-10.90
46-55	48	4.08	0.98-16.94
56-65	63	2.42	0.59-9.95
≥66	52	1.65	0.40-6.81

Smokers were at excess risk of cancers of the head and neck after modelling (OR = 1.72, 95% CI = 1.09-2.71, Table 4.6). When risk was estimated stratified by amount smoked compared to never smoked, heavy smokers had an OR of 2.28 (95% CI = 1.43-3.62). Risks were close to unity and non-significant among light and medium smokers (ORs = 0.88 and 1.22, 95% CIs = 0.51-1.53 and 0.66-2.28).

Stomach Cancer

Risk for stomach cancer was significantly lower among men with a secondary, technical or university education (Table 4.6). When the reference group was men with an elementary education only, ORs rose from 0.48 (95% CI = 0.31-0.76), among university-educated men to 0.58 (95% CI = 0.40-0.84), among those with technical education to 0.61 (95% CI = 0.42-0.87) among men with a secondary education. However, the trend was not statistically significant. Smokers had twice the risk of non-smokers (Table 4.6) and risk increased with the amount smoked. Compared to those who never smoked, ORs were 1.32 (95% CI = 0.79-2.20) for medium smokers and 2.36 (95% CI = 1.49-3.73) for heavy smokers. Being a farm owner (prestige rank V), compared to blue collar workers including farm labourers (rank VI), contributed to a deficit in risk for stomach cancer (OR = 0.58, 95% CI = 0.56-0.92). ORs were not significantly affected by the other prestige ranks, nor was there an overall trend in magnitude of risk associated with occupational prestige.

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[7] ≤30 is the baseline

Table 4.11

Smoking Among Stomach Cancer Cases

Variable	Level	Mean Lifetime Cigarettes
Education	Elementary	264,516
	Secondary	226,296
	Technical	194,984
	University	246,544
Occupational Prestige	I. Manager/Professional	219,241
	II. Semi Professional/Technicians	233,030
	III. Supervisor/Middle Management	254,945
	IV. Lower White Collar	267,686
	V. Farm Owners	241,636
	VI. Blue Collar Labourers	205,136

The smoking habits of stomach cancer cases did not follow a distinct pattern according to income or prestige level (Table 4.11), so it does not seem likely that either of these SES measures is a proxy for smoking. An examination of self-reported exposure history did not suggest any association between income or occupational prestige and exposure. There was no interaction between education, cigarette smoking and occupational prestige (Table 4.7).

Laryngeal Cancer

Not unexpectedly, the most powerful factor associated with laryngeal cancer was smoking (OR = 15.69, 95% CI = 3.88-63.52, Table 4.6). Compared to men with an elementary education only, a secondary/technical education reduced risk (OR = 0.57, 95% CI = 0.36-0.72), as did having a university education (OR = 0.48, 95% CI = 0.30-0.79), but again, the trend was not significant. Being a farm owner/operator was associated with a deficit in risk, compared to blue collar workers (OR = 0.51, 95% CI = 0.28-0.93), and the trend test was statistically significant. However, the other occupational prestige ranks were not related to risk.

As smoking exerted such a profound effect upon risk for laryngeal cancer, education and occupational prestige were investigated as possible proxy measures of smoking habits. Elementary and university educated men showed a clear progression in amount smoked, but the progression was somewhat unexpected. The percentage of men with laryngeal cancer who had never smoked increased from 2% among those with an elementary education only, to 4% among those with a technical education, to 5% among the university-educated. The pattern for heavy smoking was reversed: among men with laryngeal cancer who did smoke, heavy smoking was reported by 58% of those with an elementary education, 64% of those with secondary schooling, 65% of technically trained men and 79% of the university-educated. Mean lifetime amount smoked reflected a similar pattern; means were 288,314 cigarettes per lifetime for the elementary and

402,112 cigarettes per lifetime for the university levels. Mean cigarettes smoked over a lifetime for the secondary and technical levels were similar to that in the elementary level (293,971 and 280,431 cigarettes per lifetime respectively). As risk for laryngeal cancer increased directly with amount smoked (Table 4.12), it seems unlikely that education is a reliable indicator of smoking habits among these cases.

Table 4.12

Risk for Laryngeal Cancer By Amount Smoked

	Unadjusted	
Smoking Category[8]	Odds Ratio	95% Confidence Interval
Light	7.11	2.17-23.36
Medium	12.76	3.83-42.49
Heavy	20.16	6.38-63.66

Occupational prestige rank did not reflect smoking habits either. The pattern of amount of smoking by rank was random and mean number of cigarettes smoked per lifetime was similar for all ranks, with the exceptions of ranks IV (lower white collar, mean=363,538) and V (farm owner/operator, mean=179,784). Mean cigarettes smoked for the other prestige ranks were: I = 287,471, II = 283,799, III = 294,165, VI = 272,826. The lower mean among farm owners/operators is congruent with the deficit in risk for laryngeal cancer associated with this group in the logistic regression model, but the pattern of amount smoked is not. The proportion of non-smokers, light, medium and heavy smokers in this group was much like the patterns in the other groups. Given the foregoing, it is not surprising that there was no interaction between smoking and education or occupational prestige (Table 4.7).

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[8] Relative to never smokers

Lung Cancer

The variables modelled in lung cancer were age, smoking (dichotomized into never smoked, light smokers of < 182,626 cigarettes per lifetime, heavy smokers of ≥ 182,626 cigarettes per lifetime), imputed earned income, education and occupational prestige. The latter SES index was not associated with risk in this model, nor was age. The largest contributor to excess risk for lung cancer in the logistic regression model was cigarette smoking (Table 4.6). Heavy smokers had an OR of 5.51 (95% CI = 4.25-7.13), compared to non-smokers, while light smokers had an OR of 1.87 (95% CI = 1.41-2.48). Unadjusted risk for lung cancer approximately doubled as amount smoked increased, from light to medium to heavy, compared to risk in non-smokers (Table 4.13). The ORs for lung cancer and smoking may be lower than most reported in the literature because the cases were limited to men aged 19 to 74, and risks may be lower in this younger group than in the whole population, which contains men older than 74 as well.

Table 4.13
Risk For Lung Cancer By Amount Smoked

	Unadjusted	
Smoking Category[9]	Odds Ratio	95% Confidence Interval
Light	6.96	3.73-12.98
Medium	14.27	7.62-26.75
Heavy	29.05	15.94-52.94

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[9] Relative to 0 cigarettes

A university education contributed substantially to a deficit in lung cancer risk (OR = 0.52, 95% CI = 0.39-0.68), as did a high income (OR = 0.75, 95% CI = 0.60-0.95) and secondary/technical education (OR = 0.76, 95% CI = 0.65-0.90), but to a lesser extent (β = 0.28 and 0.27 respectively). Tests for tend showed that only income exhibited a statistically significant trend. Comparing amount smoked among men with lung cancer by educational level (Table 4.14), among the university-educated there were more non-smokers and light smokers and fewer heavy smokers. Among cases with a mean lifetime annual earned income of $20,000 or more, a larger percentage never smoked or were light smokers and a smaller percentage were medium or heavy smokers, compared to men in the other income categories.

Table 4.14

Amount Smoked By Education and Income Among Men With Lung Cancer

	Amount Smoked			
	Never	Light	Medium	Heavy
Education:	n(%)	n(%)	n(%)	n(%)
Elementary	3(1)	46(14)	40(12)	238(73)
Secondary	2(1)	24(11)	24(11)	167(77)
Technical/Trade	1(1)	24(16)	24(16)	102(68)
University	5(6)	15(18)	10(12)	54(64)
Income:				
<$10,000	3(1)	42(14)	38(12)	224(73)
$10,000-19,999	4(1)	38(13)	41(14)	203(71)
≥$20,000	2(2)	18(17)	11(10)	74(70)

The distribution of cases by amount smoked was quite similar at the secondary and technical/trade levels of education and income levels under $20,000. Therefore it appears that the SES indices education and income better represent smoking habits among lung cancer cases, at least at the extremes of amount smoked, than was the case with laryngeal cancer.

However, in addition to smoking habits, some other features of education and of income must be related to risk for lung cancer, because there were significant interactions between smoking and both SES variables (Table 4.7). The joint effects were additive; the relative excess risks of lung cancer due to the interactions of smoking and education and smoking and income were 9.14 and 11.83 respectively. The proportions of disease attributable to the interactions were 0.38 and 0.43 respectively. There was no interaction between income level and education.

Malignant Cutaneous Melanoma

Compared to men 50 years old and older, those under 50 had a substantially lower risk for malignant melanoma (OR = 0.27, 95% CI = 0.20-0.35) in the multiple logistic regression model. Although the trends were not statistically significant, higher education and higher income were both positively associated with risk. Compared to the risk estimate among cases with an elementary education, risk rose with educational level, from an OR of 1.80 (95% CI = 1.22 - 2.65, Table 4.6) at the secondary or technical level, to 2.39 (95% CI = 1.56-3.66) for university. ORs for malignant melanoma also rose with imputed earned income; OR for those earning a mean annual income of $10,000 to $19,999 was 1.64 (95% CI = 1.14-2.36) and for those earning $20,000 or more, the OR was 1.75 (95% CI = 1.17-2.61). The relationship between risk for malignant melanoma and age was rather complex. Inspection of the ORs associated with the individual variables showed increasing risk with decreasing age and increasing education and income (Table 4.15). Odds ratios for five age categories demonstrated decreasing risk with increasing age, the opposite result of the application of multiple logistic regression and two age categories. The explanation for the anomalous results may lie in the age distribution of melanoma cases compared to the control sample. Melanoma cases were similarly distributed in age, with 53% under age 50 and 47% older. Only 18% of the controls were under age 50, while 83% were older. Occupational prestige rank was not related to risk for this cancer either directly or as an effect modifier, nor was cigarette smoking. There was no trend apparent in the association between occupational prestige rank and risk for malignant melanoma.

There was no significant interaction between age and income, nor between education and income (Table 4.7). The interaction between age and education, however, was significant. The relative excess risk of disease due to the interaction was 6.48 and 77% of the disease was attributable to the interaction.

Ultraviolet light is a risk factor for malignant melanoma, so recreational sunlight exposure was also analyzed. Only 4% of the melanoma cases with elementary schooling reported recreational sunlight exposure, compared to 12% of controls at this educational level. Among cases with technical or university education, 74% reported recreational exposure, compared to 65% of controls.

Non-Melanotic Skin Cancer

There were no interactive effects on NMSC risk from any combination of SES variables (Table 4.7). In the logistic regression model, risk increased with education, with imputed earned income and with age (Table 4.6). For education and income, individual ORs demonstrated an association of increased risk until about age 55, then declining risk in the older age groups (Table 4.16). These associations showed no statistically significant trend.

Table 4.15

Risk Estimates For Malignant Melanoma

			Unadjusted	
			OR	95% CI
Age	<30	versus 31-45	0.98	0.61-1.55
		versus 46-55	0.37	0.23-0.61
		versus 56-65	0.19	0.11-0.30
		versus ≥66	0.09	0.06-0.16
Education	Elementary	versus Secondary	3.20	2.08-4.92
		versus Technical/Trade	3.55	2.32-5.45
		versus University	6.48	4.28-9.81
Income	<$10,000	versus $10,000-19,999	2.81	1.97-4.02
		versus ≥$20,000	5.49	3.87-7.80

In general, risk increased with increasing occupational prestige rank, but the trend was not significant for the truncated prestige categories. Risk was also significantly higher in farm owners, compared to blue collar labourers (Table 4.16). Disruption of the tendency for risk to increase with increasing occupational prestige is likely related to exposure to sunlight. Exposure to sunlight did not increase consistently with prestige rank (Table 4.17) but ORs associated with self-reported exposure all significantly exceeded one, at every rank, when the base line reference group consisted of people who reported no exposure. Other exposures were also associated with excess risk of NMSC. The ORs associated with self-reported exposure to video display terminals and fluorescent lights were significantly high, compared to no exposure, at every prestige rank. ORs for exposure to natural gas and welding fumes were elevated at most of the occupational prestige levels. However, there was no pattern between risk estimate change and prestige rank for any of these exposures (Table 4.18).

Prostatic Cancer

Age was the best predictor of risk for prostatic cancer, with men over the age of 70 at nearly twice the risk, compared to men under 70. An earned income over $10,000.00 was protective and the trend for risk to decrease with increasing income was significant. Any post-elementary education conferred some protection, with significant deficits remaining after logistic regression was applied (Table 4.6). Risk for prostatic cancer was not affected by any interaction between age, income and education.

Testicular Cancer

In marked contrast to prostatic cancer, deficits in testicular cancer were associated with men over the age of 30. Employment in one of the higher ranked occupational status categories also was protective in the logistic regression (Table 4.6). Increase in imputed average annual lifetime earned income contributed directly to significantly increased risk, with ORs increasing from 3.75 at \$10,000-19,999 to 12.03 at ≥\$20,000. Compared to men with elementary education, those with secondary or technical education were at about 50% greater risk, while risk for those with university education more than doubled (Table 4.6).

Table 4.16

Risk Estimates For Non-Melanotic Skin Cancer

		Unadjusted	
		Odds Ratio	95% C.I.
Education	Elementary versus Secondary	1.65	1.38-1.97
	versus Technical	1.83	1.53-2.19
	versus University	2.63	2.19-3.16
Income	<10,000 versus 10,000-19,999	1.32	1.14-1.52
	versus ≥20,000	1.51	1.29-1.77
Age	<30 versus 31-45	4.24	2.12-8.47
	versus 46-55	5.14	2.59-10.10
	versus 56-65	4.04	2.05-7.96
	versus >65	4.12	2.10-8.12
Occupational Prestige	Ranks VI versus I	2.54	2.09-3.09
	versus II	2.49	1.83-3.40
	versus III	1.90	1.52-2.37
	versus IV	1.81	1.50-2.19
	versus V	2.14	1.73-2.65

However, risk for testicular cancer did not show significant trends associated with any of the SES indices. Although it would seem reasonable that age, education, income and occupational prestige would be closely related, neither the correlation matrix (Table 4.4) nor the estimation of joint effects confirm this. There was no interaction between these variables in testicular cancer risk.

Table 4.17
Risk for NMSC Associated With Exposure to Ultraviolet Light by Prestige Rank

			Unadjusted	
Prestige Rank	% Exposed Cases	% Exposed Controls	Odds Ratio	95% CI
I. Managerial/Professional	16	6	3.07	2.01-4.68
II. Semi Professional/ Technical	19	10	2.14	1.01-4.55
III. Supervisory/Middle Management	12	4	3.07	1.69-5.57
IV. Lower White Collar	3	6	2.52	1.70-3.73
V. Farm Owner	14	6	2.42	1.45-4.01
VI. Blue Collar	9	4	2.63	1.51-4.57

Bladder Cancer

Only two variables were associated with bladder cancer. Risk was about twice as high in men who smoked, compared to never smokers. Income affected the risk estimate less than did smoking and in the opposite direction. Men with earned incomes ≥$20,000 had a significant deficit in risk, compared to those earning less than $10,000 (Table 4.6). Again there was no interactive effect on risk nor was there a significant trend associated with income.

Table 4.18
Statistically Significant Risk Estimates Associated With Other Exposures by Prestige Rank in NMSC

						Unadjusted	
	Rank	Exposed Cases		Exposed Controls		Odds Ratio	95% Confidence Interval
		n	%	n	%		
Video Display Terminals	I	67	23	97	11	2.42	1.72-3.42
	II	24	35	28	13	3.57	1.89-6.74
	III	26	16	38	6	3.13	1.84-5.32
	IV	32	10	76	5	1.88	1.22-2.90
	V	9	5	13	2	2.58	1.09-6.13
	VI	11	5	24	2	3.69	1.78-7.65
Fluorescent Light	I	178	60	330	37	2.60	1.99-3.40
	II	40	59	69	33	2.94	1.68-5.16
	III	96	58	185	27	3.66	2.57-5.19
	IV	153	45	386	27	2.25	1.76-2.87
	V	57	29	150	21	1.53	1.07-2.18
	VI	70	34	200	13	3.59	2.59-4.96
Natural Gas	I	28	9	43	5	2.08	1.27-3.42
	II	8	12	8	4	3.38	1.22-9.40
	III	27	16	50	7	2.44	1.48-4.03
	IV	38	11	105	7	1.62	1.09-2.39
	VI	20	10	87	5	1.86	1.12-3.10
Welding Fumes	III	33	20	53	8	2.93	1.82-4.70
	IV	60	18	189	13	1.43	1.04-1.97
	V	64	32	165	23	1.60	1.13-2.25
	VI	27	13	112	7	2.00	1.28-3.12

Brain Cancer and Hodgkin's Disease

Age and imputed earned income were related to risk for brain cancer and for Hodgkin's disease; the relations were in the same direction. Men over 50 were at greatly reduced risk of brain cancer, while those over 35 enjoyed a marked deficit in risk for Hodgkin's disease. Risk for brain cancer increased from 1.77 at an income level of $10,000 to 19,999, to 1.95 at an income over $20,000, compared to an income less than $10,000 and the trend was statistically significant. The OR for Hodgkin's disease was 1.73 at an income exceeding $20,000 (Table 4.6), but overall there was no trend in risk. Estimation of the interaction showed there were no joint effects at either site (Table 4.7).

Other Cancer Sites

Risk for non-Hodgkin's lymphoma, leukaemia and thyroid cancer decreased as age increased (Table 4.6). There was no association between these cancers and any of the SES indices, nor with smoking or alcohol consumption. Cancer at several sites was significantly associated with one or more SES indices when unadjusted ORs were estimated (Table 4.19), but the effects were reduced to non-significance after control for age (≤ 50, 51-69, ≥ 70), with one exception. The significant deficit in risk for kidney cancer among men with a secondary education was unaffected by age, but was reduced to non-significance by control for smoking (never smoked, light smoker, heavy smoker). The remaining cancer sites were not associated with any SES indices or were too few in number for meaningful analysis.

Table 4.19

Risk Estimates for Other Cancers

Cancer	SES Index	Cases	Unadjusted		Stratified	
			Odds Ratio	95% Confidence Interval	Odds Ratio	95% Confidence Interval
Colon	Income 10,000-19,999	251	0.81	0.68-0.97	0.84	0.70-1.02
	≥20,000	138	0.68	0.55-0.85	0.96	0.76-1.22
	Education University	100	0.71	0.56-0.91	0.87	0.68-1.13
	Prestige Level II	18	0.58	0.35-0.95	0.67	0.40-1.12
Pancreatic	Income ≥20,000	7	0.38	0.17-0.87	0.46	0.17-1.22
Bladder	Income ≥20,000	87	0.67	0.52-0.87	0.80	0.59-1.09
	Education University	62	0.66	0.49-0.90	0.75	0.55-1.02
	Prestige Level II	12	0.54	0.29-0.98	0.61	0.33-1.12
Testicular	Income 10,000-19,999	36	8.93	3.17-25.13	2.13	0.76-5.94
	Education Technical	40	6.15	2.98-12.71	1.72	0.82-3.64
	University	42	8.79	4.26-18.11	1.63	0.76-3.50
	Prestige Level V	3	0.14	0.04-0.47	0.36	0.12-1.14

Table 4.19 (cont'd)

Risk Estimates for Other Cancers

Cancer	SES Index	Cases	Unadjusted		Stratified	
			Odds Ratio	95% Confidence Interval	Odds Ratio	95% Confidence Interval
Kidney	Income 10,000-19,999	120	1.37	1.03-1.81	1.16	0.84-1.58[1]
	Education Secondary	51	0.67	0.48-0.95	0.71	0.50-1.02[2]
	Prestige Level I	41	0.67	0.46-0.98	0.69	0.47-1.00[3]
	Prestige Level III	25	0.58	0.37-0.91	0.64	0.41-1.00[2]
Brain	Income 10,000-19,999	52	2.72	1.60-4.61	1.47	0.79-2.73
	Education Secondary	36	2.08	1.22-3.55	1.24	0.68-2.26
	Technical	43	2.69	1.61-4.52	1.39	0.76-2.52
	University	25	2.10	1.18-3.73	0.99	0.51-1.95
Non-Hodgkin's Lymphoma	Income 10,000-19,999	106	1.55	1.14-2.11	1.15	0.82-1.61
	Education Secondary	70	1.57	1.10-2.24	1.34	0.93-1.92

[1] Stratified by age

[2] Stratified by smoking (never, light and medium, heavy)

[3] Stratified by alcohol (yes/no)

Table 4.19 (cont'd)
Risk Estimates for Other Cancer Sites

Cancer Site	SES Index	Cases	Unadjusted		Stratified	
			Odds Ratio	95% Confidence Interval	Odds Ratio	95% Confidence Interval
Hodgkin's Disease	Income 10,000-19,999	32	2.63	1.35-5.12	1.42	0.63-3.21
	Education Technical	33	2.66	1.48-4.80	1.22	0.64-2.32
	University	32	3.50	1.94-6.33	1.24	0.63-2.43
Leukaemia	Income 10,000-19,999	66	1.55	1.05-2.30	0.97	0.63-1.47
	≥20,000	54	1.99	1.32-2.99	1.24	0.80-1.94
Thyroid	Income 10,000-19,999	14	4.59	1.32-16.00	3.06	0.73-12.87
	≥20,000	9	4.59	1.24-16.98	2.51	0.37-17.27
	Education Secondary	9	5.69	1.23-26.37	3.57	0.65-19.60
	University	6	5.49	1.11-27.25	3.85	0.57-26.00
Bone and Connective Tissue	Income ≥20,000	29	2.98	1.59-5.57	1.65	0.78-3.53
	Education Secondary	22	2.15	1.08-4.27	1.90	0.91-3.97
	University	16	2.26	1.08-4.72	1.41	0.49-4.03
Oesophageal	Education Secondary	5	0.35	0.13-0.94	0.40	0.15-1.06

CHAPTER V

DISCUSSION

A. CONSISTENCY OF SES - CANCER RISK ASSOCIATIONS

The most effective way of controlling cancer is to prevent the disease. To plan appropriate interventions and reduce the prevalence of risk factors for cancer among people, it is important that groups at high risk can be efficiently identified. The primary objective of this analysis was to establish if education, earned income or occupational prestige level exhibited sufficiently consistent associations with cancers at specific body sites to permit the use of one or more of these SES indices as an identifier of high risk groups. For maximum efficiency, one index is preferred over several; therefore a second objective was to determine which of the three indices is the best predictor of cancer risk in Alberta. Finally, it is important to understand the nature of any associations and their operation as direct indicators of factors which determine cancer risk or as confounders or effect modifiers.

In this case-control analysis of data from Alberta men, all three indices of SES proved to be significantly associated with risk at only three out of 26 cancer sites, after control for age at diagnosis (which also controlled for age at first employment), cigarette smoking and alcohol consumption. This suggests that a composite SES measure which combines income, education and occupational prestige level may be too non-specific to be appropriate. Risk for lip cancer decreased directly as education, occupational prestige and imputed mean annual earned income increased. Risk for testicular cancer increased as income and education increased, exhibiting a non-significant dose-response pattern, but was significantly lower in upper occupational prestige ranks, compared to lower ranks. Upper occupational prestige ranks are assumed to be associated with higher incomes and education levels, so these findings are unexpected. Risk for NMSC also increased with both education and income and was significantly low in all occupational prestige ranks, compared to rank VI, again somewhat contradictory.

Age alone was a predictor of risk for non-Hodgkin's lymphoma, leukaemia and thyroid cancer. None of the SES indices were significantly related to these cancers, nor was cigarette smoking. The latter has been implicated as a risk factor for leukaemia in several recent studies elsewhere, but not in Alberta.[113-116] A pattern of increasing risk with an increase in the number of cigarettes smoked has also been demonstrated elsewhere,[117-119] but not conclusively.[120,121] The inconsistency of the associations between the SES indices and these cancers suggest that income, education and occupational prestige level are indicators of behaviours which affect risk, but not uniformly.

B. CONSISTENCY OF INCOME - CANCER RISK ASSOCIATIONS

Consideration of the individual SES indices showed income was significantly associated with risk for nine out of 26 cancer sites. In addition to the risks for lip and testicular cancers and NMSC discussed in section A, increased earned income was associated with significant decreases in risk for cancers of the lung, prostate and bladder. The low risk for lung cancer is consistent with other studies,[22] but there do not appear to be any published reports on lip or bladder cancer and SES. Cigarette smoking is a demonstrated risk factor for both lung and bladder cancers[122] and may be a factor in lip cancer.[107] Re-examination of the logistic regression models suggests that smoking was the most important factor affecting lung cancer risk, while only an income of ≥$20,000 was statistically significant. Although income appeared to explain all of the variance and the trend for risk to decrease as income increased was statistically significant, incomes below the top category were not predictive of lung cancer risk. This is reflected by distribution of smokers by income - among men earning <$10,000 and also among those earning $10,000-19,999, 21% were heavy smokers compared to 8% among men earning ≥$20,000. The correlation between income and smoking was also relatively low (Table 4.4), suggesting that even though smoking is highly predictive, income alone is probably not.

Ever smoking versus never smoking was a powerful predictor of excess risk for bladder cancer, while only the highest income category was a useful predictor. Sparsity of data prevented analysis by more refined smoking categories, as it prevented trend analysis.

For lip cancer, income was again not a strong factor, with only the top category predicting significantly lower risk. Smoking was dropped from the regression model altogether, suggesting its effects do not alter risk substantially. Stratification analysis supported this, as the ORs did not change when they were adjusted for smoking.

Alcohol consumption was not a factor in any of the analyses.

As well as a weak surrogate for smoking habits, income may be a surrogate for diet.[123] Bladder cancer, for example, is epithelial in origin and is lower among people whose diet is high in fruit or vegetable consumption.[124] The effects of diet could not be assessed because this variable was not included in the study, in the interests of brevity.

Imputed earned income was also predictive of increased risk for malignant cutaneous melanoma, testicular and brain cancers, with risk increasing directly with increasing income, although this trend was statistically significant for brain cancer risk only. Income explained 97 to 100% of the variance; however, the underlying determinants for which income is a proxy are not clear. Increased risk for cutaneous malignant melanoma has been related to intense, intermittent exposure to sunlight.[125,126] The site distribution argues for this hypothesis, as incidence is highest in least-exposed areas of the body.[127] High income earners have the resources necessary to allow vacation activities in sunny climates and the suggestion is that the recreational and social activities of high income earners increase the likelihood that severe, acute exposure to sunlight on normally covered, untanned areas of the skin would occur, resulting in actinic DNA damage and pathogenesis leading to cutaneous malignant melanoma.[128]

The Alberta finding that risk for testicular cancer increases directly with earned income is consistent with reports of elevated risks among men in upper social classes.[22,27,35,129] Clearly age is strongly related to risk for testicular cancer, remaining significant after modelling, as well as markedly altering the OR in age-stratified analysis, from an unadjusted OR for 8.93 (95% CI = 3.17 - 25.13) to an age-stratified OR of 2.13 (95% CI = 0.76 - 5.94, non-significant). In addition, although age and income were not highly correlated (Table 4.4) younger men tended to be high income earners, with 73% earning ≥$20,000 and only 3% earning <$10,000. As the majority of testicular cancer cases are young men (60% under age 35, 93% under age 50), it appears that income is at least partially a surrogate for the effect modifier, age. Income was positively related to and significantly predictive of risk for cancer of the brain, a finding consistent with other reports linking increased brain cancer risk with upper socioeconomic status.[130,131]

A possible explanation for the brain cancer-earned income link may be detection bias. People in the lowest income bracket may be less conveniently located to take advantage of improved brain - imaging techniques, such as computed tomography and magnetic resonance imaging, normally situated in large urban hospitals.[132] Those in the higher income brackets may have readier access to specialists, also concentrated in urban, higher income areas. Another factor which may have affected the results for income is that risk has been shown to be elevated in a number of professional occupational groups with high incomes. These pathologists, dentists, chemists and other workers in the medical profession are exposed on the job to a variety of things which have been implicated in brain cancer, including relatively high levels of ionizing and non-ionizing radiation, infection, mercury, formaldehyde and other laboratory chemicals.[131,133-134] As with testicular cancer, there was a strong association between young age and high income, which increased the predictive ability of income, as brain cancer cases also tend to be younger (60% under age 50 in the Alberta series). Therefore, income in brain cancer could be a surrogate for detection bias, occupational exposure or age. A high income was also associated with high risk for Hodgkin's disease. Previously, only one report has linked risk for Hodgkin's disease with income.[135] The factors which affect risk for Hodgkin's disease have not been clearly demonstrated, but this cancer follows much the same pattern as does testicular cancer. The epidemiology of both diseases is quite similar, with bimodal age distributions, similar links with SES and few specific occupational associations. It has been argued that risk for both diseases is determined by factors affecting early childhood or even by prenatal factors.[35] Although it is not clear what these factors could be, one suggestion is that exogenously administered hormones in early pregnancy for the control of nausea may affect the incidence of cryptorchidism, which increases risk for testicular cancer.[136] It seems reasonable that this type of drug use would be more common among upper-income level women, compared to women in the lowest income bracket. Parental occupation may be a factor[25] in both diseases through the medium of job exposure or diet, both of which can be related to endogenous hormone production and thus to risk for these cancers.[136] Thus, the predictive ability of income for testicular cancer and Hodgkin's disease risks in the Alberta data may result from the covariation of income with other lifestyle factors, rather than from income alone.

NMSC risk was imperfectly predicted by income, with high risk significantly related to income >$10,000 only, leaving 46% of the variance unexplained. Older age and chronic, cumulative sun exposure have been associated with NMSC[137-139], but both factors would be expected to be common among the lower income bracket. Income does not appear to be a surrogate for either of these factors in this case.

Income >$10,000 was also predictive, for low risk of prostatic cancer. Risk decreased significantly as income increased and income variation was responsible for most of the variation in the ORs (Figure 10). It is somewhat paradoxical that increasing age is one of the few clearly demonstrated risk factors for prostatic cancer,[140-144] yet in this data base, risk decreased as income increased and high income was related to young age, not to advanced age - 66% of men aged 65 and over had incomes of <$10,000 per year.

In summary, the Alberta data showed a reasonable degree of consistency between imputed earned income and risk for cutaneous malignant melanoma, testicular cancer, prostatic cancer and brain cancer. Income could be a useful and readily available index for identifying people at high risk for these cancers. Hodgkin's disease was not strongly associated with income. Both prostatic cancer and Hodgkin's disease were strongly related to age, which is readily available from provincial health care enrolment lists. Age would seem to be more useful than income for identifying people at high risk for these cancers. This analysis of income did not suggest that people at high risk of NMSC could be identified consistently enough to be useful.

C. CONSISTENCY OF EDUCATION - CANCER RISK ASSOCIATIONS

Education was predictive of risk for eight cancer sites after modelling. Decreases in risk were significantly associated with increases in education for cancers of the stomach, lung, larynx and prostate gland, as well as lip. However, none of these exhibited statistically significant trends. The Alberta results corroborate the few results from other countries relating to these cancer sites,[22,26,29,30-32] with the exceptions of prostatic and lip cancers. Increased risks for prostatic cancer have been significantly associated with increased education both in Finland[26] and in a previous Alberta study.[145]

The explanation for these anomalous results may be marked age differences between educational levels. Fully 20% of the prostatic cancer cases with only an elementary education were over 70 years of age, while only 5% with university were in this age group. As prostatic cancer is strongly associated with increasing age, the decreased risk at higher educational levels may be the result of a disproportionate frequency of cases over 70 and with elementary education. Possibly dichotomization of age in modelling was insufficient to control for such a large disparity in age distribution. When stratified analysis was applied, controlling for age increased risks for prostatic cancer, 32% for men with a secondary education, 43% for those with a technical education and 83% for the university-educated (although the ORs remained significantly below unity).

The predictive value of educational level in lung cancer was as an indicator of cigarette smoking habits, which were more strongly associated with lung cancer risk than education in the logistic regression model. The interaction of education level and smoking habits may have introduced some bias, which might tend to reduce the differences in ORs by education. There were more men with a university education who had never smoked and fewer heavy smokers, both of which would reduce risk for lung cancer, compared to men with an elementary education. However, among light and medium smokers, more men with university education smoked more cigarettes, compared to men with an elementary education. The net effect may have been to increase risk among the university educated to a small extent.

Decreased risk for lip cancer in higher educational categories has not previously been reported. A study in the United States[36] found no relation between education or other SES measures and lip cancer risk. The Alberta finding is not inconsistent with reported knowledge of the epidemiology of lip cancer, however, if education is a proxy for occupation. Rates are notably high in farmers and fishermen,[111] who may not require high levels of formal education in their professions. Among farmers in this data set, 59% had only an elementary education.

Testicular cancer, malignant melanoma and NMSC increased with education, results consistent with other reports of elevated risks for these cancers in upper social classes.[12,17,25,113] The elevated risk for testicular cancer was partially a result of the education level distribution by age, coupled with the fact that testicular cancer occurs most commonly in men under 40 years of age. Thirty-seven percent of men with cancer in Alberta under the age of 40 were university educated, compared to 5% with an elementary education. This SES index also appears to be a surrogate for age as a factor in testicular cancer, as was the case with income. The significant increases in risk for cutaneous malignant melanoma with increased education may also have reflected the covariation of this index with age.

For malignant melanoma, application of Rothman's method for assessing interaction in multivariate models [96] showed that age and education interacted significantly. When the age distribution of malignant melanoma cases is considered, risk increases dramatically to about age 45; under age 45, about 30% of cases were university-educated and 5% had elementary education only. Risk drops in the middle years and rises again, although not so precipitously, after age 50. The rise in risk after age 55 is parallelled by a drop in the proportion of cases with university education (14%)

and an increase in the proportion with elementary education only (33%). The result is a high risk of malignant melanoma associated with both the highest and the lowest education levels. This underscores that education per se is not a determinant of malignant melanoma, but a reflector of a determinant or determinants. For example, people with advanced education more often have indoor than outdoor jobs, and relatively high levels of exposure to fluorescent lighting. Current opinion, based on previous epidemiological, clinical and animal studies, is that fluorescent light exposure is a potential risk factor for malignant melanoma.[146] The results for NMSC are somewhat difficult to interpret. Most of the epidemiological evidence suggests that it is the cumulative effect of chronic, low intensity sunlight exposure which affects risk for NMSC rather than acute intermittent exposure which increases risk for malignant melanoma. In the Alberta data, risk for NMSC was positively related to education and it is somewhat of a paradox that both melanotic and non-melanotic skin cancer have the same relation to this index, as the aetiologies differ. Also, chronic low dose exposure appears to be protective against cutaneous malignant melanoma,[147] either through increased melanin production,[128] which thickens the skin and reduces depth of exposure, or through increased vitamin D production, which inhibits the melanotic cells.[132] Based on this, it would be expected that among people with high risk for NMSC, risk for malignant melanoma would be low or intermediate.

It is possible that education among NMSC cases is a surrogate for screening behaviour, rather than sunlight exposure. Most NMSC cases do not report to the provincial cancer clinics for either diagnosis or treatment and become registered only through pathology reports, which lack contact information (address, telephone). The cases included in this analysis are mostly people who did report to a cancer clinic, probably for screening. These NMSC cases may not be representative of all cases, but a self-selected sample with greater awareness of symptomatology and the importance of early detection of cancer. Greater awareness has been shown to be more common among people with advanced education - for example, participants in a skin cancer screening clinic in Connecticut were all high school or college educated.[148] As was the case for income, education may also be a surrogate for diet. A low level of education has been associated with lower consumption of fresh fruit and green vegetables.[124] High consumption of fruit and vegetables has in turn been associated consistently, although not universally, with reduced risk for cancer at most sites.[123] This is particularly the case for epithelial cancers. The decrease in risk with increased education for epithelial cancers of the lung, larynx and stomach may be a result of diet, rather than of education per se.

In summary, although education was significantly associated with cancer risk at a number of sites in modelling, analysis of the trends in risk change with education level were all non-significant. The results suggest that education level has little utility as an identifier of high risk groups for any cancer.

D. CONSISTENCY OF COLLAPSED OCCUPATIONAL PRESTIGE - CANCER RISK ASSOCIATIONS

Collapsed occupational prestige level was a significant factor affecting risk in the logistic regression analysis for six cancers, but trend analysis showed that only risk for laryngeal cancer was statistically significant. Risk increased as occupational prestige decreased. However, when all the analyses are considered, it is clear that the significant trend is based on a significantly decreased risk among farm owners compared to blue collar workers and that prestige level was the least important variable in the regression model. Therefore interpretation of the significant trend is difficult and it would seem that occupational prestige level was not a reliable predictor of risk, regardless of statistical significance. Smoking was clearly associated with risk for laryngeal cancer (Table 4.6). Scrutiny of smoking rates in farm owners/operators compared to blue collar workers (rank V versus VI), suggested that prestige level may be a proxy for cigarette smoking. Twenty-six percent of farm owners and operators never smoked, compared to 14% of blue collar workers.

In general, the Alberta data suggest that income may be a useful indicator of cancer risk at some sites, but that education and occupational prestige are not. The data also suggest there would be no particular advantage in using an SES index which combines income, education and occupational prestige level. For most sites, superimposing the trends for the three separate indices showed that there was differential divergence between them at the upper and lower ends of the SES scales. Therefore, a combined SES index would be meaningless except in the middle range and not usefully predictive. The exceptions were the SES trends for cancers of the lip, lung and prostate, which either coincided or were parallel. However, only income demonstrated largely statistically significant trends, while education and occupational prestige did not. The exception was lip cancer, for which trends in risk were the same, although not statistically significant for any of the three SES indices (p=0.07, 0.07 and 0.08 for income, education and occupational prestige respectively). It is more efficient to use one index, rather than a combination of three, if the predictive value is the same.

E. SES INDICES AS SURROGATES FOR DISEASE DETERMINANTS

SES is assumed to be a surrogate for behaviours and environments that are direct determinants of disease. Imputed earned income, education and occupational prestige level were selected as indices of SES for this study primarily because they represent both social and economic facets of life. A further assumption was that direct disease determinants are influenced by both social and economic factors, rather than by economic factors alone, the focus of social class analysis. It was hypothesized that the SES indices selected would be associated with direct disease determinants in a systematic way and that this would be reflected in the risk estimates relating likelihood of disease and SES index. These consistent patterns would permit the identification of high risk groups in the general population through the use of readily available census statistics on income, education level and employment. As it is unlikely that education or income per se are causal, they appeared to be surrogates for direct disease determinants at a limited number of cancer sites. Education was a proxy for causal or protective factors for cancers of the lip, prostate, lung and for NMSC and malignant melanoma. Income appeared to be a proxy for these factors in testicular and prostate cancers. It was speculated previously that the direct determinant in prostatic and testicular cancers might be hormonal, triggered by some aspect of diet or drug use which is different among high income/education groups compared to low income/education groups. Among people at high risk for NMSC and malignant melanoma, exposure to ultraviolet light and perhaps fluorescent light may be direct determinants, for which income and education are surrogates. Data on diet and drug use are not available for this group and exposure data are incomplete and not suitable for analysis.

Occupational prestige level appeared to represent a direct determinant or determinants of laryngeal cancer, but what these are remains unknown. Again, the SES measure may be a proxy for workplace exposures. For the other cancer sites, occupational prestige was not meaningful and may have been a confounder, related to both disease and job exposure in an undetermined way.

The interactions between smoking and earned income and smoking and education among lung cancer cases which increased risk multiplicatively, likely represent a constellation of factors associated with low income and low education. Workplace exposures to contaminants, residential air pollution and a diet low in vitamin C are possibilities which would exacerbate the effects of cigarette smoking. The interaction between youth and post-secondary education which increased risk among men with malignant melanoma may be a function of increased opportunity for recreational sun exposure among the better educated and increased susceptibility to actinic skin changes at younger ages. Age and cigarette smoking also appeared to be the only real effect modifiers, which was clear in stratified analysis. Age alone was a modifier in malignant melanoma, NMSC, non-Hodgkin's lymphoma and Hodgkin's disease and in cancers of the lip, prostate, testis and brain. Smoking alone increased risk in laryngeal and stomach cancers and both together were effect modifiers in lung cancer.

F. STRENGTHS OF STUDY DESIGN AND POSSIBLE LIMITATIONS

There are a number of factors which must be considered in assessing the Alberta results. First, histological confirmation was very high for all cancer cases (in excess of 95%). Ascertainment of cancer in the province is virtually complete, as any pathology reports dealing with malignancy must be registered under the Alberta Cancer Act. Bias due to under ascertainment or misclassification of disease was therefore not a problem in this study. Response bias was minimized, as demonstrated by a response rate of 70% or better for cancer sites shown to be related to any SES index and response averaged over all sites was 71%. Response was necessarily related to survival, with better response rates among people with cancers associated with survival times of 6 months or better. Survival has been shown to be associated with a number of indices of SES for cancers with a good prognosis, while for those associated with a poor prognosis, survival is independent of SES.[149-151] This may be a function of stage of disease at diagnosis, as cancers associated with poor survival are difficult to diagnose. For this reason, the disease may be more advanced at detection and survival largely independent of factors other than stage and extent of the cancer.

Recall bias was minimized by using cancer controls rather than population controls. Equally accurate information should be obtained from both cases and controls because recollection of past exposures should be similar. Recall among cancer cases, compared to that of population controls, may be highly influenced by their disease status.[152]

A further advantage in this study was the use of annual income, averaged over a lifetime, rather than latest income, which may not approximate lifetime income well. Estimating income from census data related to job title may be more valid than requesting income information from the individual, as self-reports may tend to inflate one's level of income for reasons of social acceptability. Also, cancer at most sites has a long induction period and current income may have little bearing on risk. Income is also a sensitive issue for most people and refusals are common when data are requested. The use of three separate indices of SES had a positive effect. The indices were not perfectly correlated, indicating each was measuring a different dimension. Use of the three indices permitted assessment of their individual effects. The impact on risk of a combined measure would have been less than was the case with the individual measures and any effects may have missed detection.

There were also some limitations. Under-ascertainment of cases may have influenced the results associated with NMSC, risk for which increased with increases in all three SES indices, against expectations. There is some indication that the men in this study with NMSC may not be representative of all NMSC cases, in that they attended a cancer facility for diagnosis. The relationship between excess risk and high SES may have been typical of this group, but not of NMSC cases who did not report to a cancer facility and who could not be contacted for participation in the study because of lack of information. Results for other cancer sites are consistent with expectations based on existing knowledge, which suggests response bias was adequately controlled for all other sites.

A disadvantage of using cancer controls rather than population controls is that the results may be confounded by a factor or factors common to cases and controls because of their disease status. The effect would be to obscure real differences and to deflect the results towards the null hypothesis. A second drawback to comparing risk factors between cancer sites, rather than between cases and population controls, is that factor prevalence may differ between the general population and cases. For example, 19% of the cancer cases and cancer controls in this study had never smoked cigarettes, compared to 24% never smokers among Canadian men generally.[153] If there is not enough contrast in the prevalence of risk or protective factors between cases and controls, the outcome would again be biased towards the null hypothesis.

Selection of the 16 rank Pineo-Porter occupational prestige classification system was a limitation because of sample size. The numbers of each level were too small to permit reliable analysis. The 6-level scale, on the other hand, may have been too non-specific to be a useful index of SES. A related problem concerned the statistical analysis in relation to sample size. Conventional chi square tests for homogeneity of the ORs could not be applied because there were too many zero cells, when the covariates age, cigarette smoking and alcohol were included with each separate SES index. Similar regression models were fitted by least-squares and t-tests for trend were applied instead.[98] Sparsity of data created problems of non-convergence for rare cancer sites where some observations were zero (for example, brain cancer), and trend tests could not be done. For this reason, interpretation of the data was based on the multiple logistic regression analysis (Table 4.6), rather than tests for trend.[96,154]

Small numbers also prevented analysis by morphology, which would have been useful. The major histological types in NMSC, for example, are squamous cell and basal cell. They differ markedly in natural history and site distribution. Although both arise from the epidermis, squamous cell carcinoma most commonly arises from pre-existing actinic or solar keratoses, exhibits significant squamous differentiation and keratin production and has a tendency to metastasize to the lymph nodes. Basal cell carcinoma has little tendency to differentiate and rarely metastasizes.[155] Squamous cell carcinoma occurs more commonly in the upper limb, compared to basal cell; both types occur with approximately equal frequency on the head and neck. The relative excess of squamous cell carcinoma on the upper limb suggests that other exposures, perhaps to chemicals, may increase risk.[156] If chemical exposure is important in the etiology of squamous but not basal cell carcinoma and exposure is associated with any or all of the SES indices used in this study, the results may have been biased towards the null hypothesis.

Another limitation was that the method used to establish income estimated earned income only. No information on income additional to job earnings was available and it is not clear how this might affect the results. Also, although the analyses were controlled for age, they were not controlled for age at first employment, which may have biased the income analyses to a small extent. A final caution to be considered is possible spurious results due to multiple testing. In any project of this magnitude, the frequency of results which are significant by chance is high. With 26 cancer sites, three SES indices and three covariates, at a 95% confidence interval, 12 of the statistically significant outcomes could be totally in error and a result of chance. This problem can be approached by making the statistical test more stringent or by adjusting the p-values according to the number of comparisons made. However, there is little to be gained by decreasing the number of false positives (type I error)[94] by increasing the number of false negatives (type II error). According to Rothman[96] broadening the confidence interval also serves no purpose if it depends on the number of comparisons made but does not relate to the effect being investigated.

G. CONCEPTUAL BASIS FOR SES

The results of this analysis contributed nothing to the question of the conceptual basis of SES. Earned income was most frequently predictive of risk, but was significantly associated with outcome for only 35% of the cancer sites investigated. If income is affected by ill health and reflects a process of social selection and if income uniquely predicts cancer at a specific site, then social selection would seem to be important. Education is more likely to affect health than be affected by it and is more likely an indicator of social causation. Multiple logistic regression analysis showed education was a predictor for risk for 31% of the cancers investigated. However, risk for 23% of the sites was predicted by both income and education, while all three SES indices predicted risk for 12% of the cancer sites. These results do not clearly support either hypothesis exclusively.

CHAPTER VI

CONCLUSIONS

It is generally agreed that SES is a risk indicator for many diseases, including cancer.[38,157] There is also a consensus that SES is not a direct risk factor, but a surrogate for other factors determined by components of SES, such as education, income, occupation, and so on, which influence carcinogenesis through lifestyle, personal behaviours, workplace and residential environments. Most previous research on SES has examined cardiovascular disease, mental disorder and diabetes mellitus; there has been a paucity of research on SES related to cancer.

The purpose of the present analysis was to clarify this question and to increase our ability to identify groups at high risk for specific cancers, on the basis of SES; data concerning many components of SES are not readily available from existing population files. Interventions could then be planned, oriented to the particular needs of the target risk group.

The first objective of the study was to determine whether the three indices selected to measure SES were significantly associated with cancer at all sites or specific sites. All three indicators were related to risk for cancer at only three sites: lip, testis and non-melanotic skin. Of twenty-six cancer sites investigated, 58% proved to be independent of these indicators. Based on these results, use of a composite SES index to identify high risk groups is not justified.

A second objective was to determine which of the indicators of SES was the better predictor. In terms of number of sites, imputed earned income, followed by education, predicted the largest number. In terms of numbers of cancer cases, income and education were strongly associated with cancer sites at which about 54% of the total number of cases occurred. Assessment of the public health impact of any disease should take into account the numbers affected and the severity of the disease. Lung cancer, with a 5-year survival rate of only 10% in Alberta,[158] was closely associated with low levels of income and education. A low level of education was related to increased risk for stomach cancer, with a 5-year survival rate of 16%. On the other hand, excess risk for brain cancer, with a 5-year survival rate of 28%, was associated with high income. The remaining cancer sites for which risk was predicted by income, education, or both, have 5-year survivals of 50% or better. Those cancer sites associated with all three SES measures have the best survival: at 5 years, 77% for lip cancer, 82% for NMSC, 79% for cutaneous malignant melanoma and 93% for testicular cancer .

Successful application of interventions in public health depends upon identification of high risk groups, but also upon knowledge of risk determinants. The analysis of the Alberta data justifies an intervention to target low income earners in the province with programmes aimed at reduction of smoking prevalence, to reduce the incidence of lung cancer. The intervention should include promotion of a healthy diet, emphasizing adequate consumption of vegetables and fruits, which has been related to reduced lung cancer risk.[159] In the absence of sub-group specific population data on tobacco use, diet or environmental air contamination, low income would seem to be a practical indicator to identify people most in need of intervention. Promotion of non-smoking and smoking cessation could also reduce the incidence of bladder and laryngeal cancers and, to a lesser extent, the incidence of stomach cancer in this group. If limited resources prevent universal programs directed at the general population, screening for the early detection of cancers of the lung, larynx and bladder should be concentrated among low income people. The Alberta analysis also suggests that men at high risk of cutaneous malignant melanoma can be identified, as those in the highest two thirds of the province's earned income distribution. Interventions to reduce risk for malignant melanoma by avoiding acute, intermittent exposure to ultra violet light should be directed at these men. A recent publication even suggests that the scientific literature supports advising the public to seek regular, modest sun exposure to reduce risk for malignant melanoma and to lower cancer mortality generally.[132] The programme might include a recommendation to reduce exposure to ultraviolet B from fluorescent lighting by using filters on workplace and domestic fixtures. Although the evidence implicating ultraviolet B as an important risk factor in malignant melanoma is not beyond question, installing light filters is a non-invasive and inexpensive measure which is unlikely to create problems and which may help reduce malignant melanoma incidence.[146] Other risk factors for cutaneous malignant melanoma are fair phenotype and severe sunburn in childhood. There are already a number of programmes in Alberta,[160,161] oriented towards reducing acute exposure to ultraviolet light among these susceptible groups, although these interventions are aimed at the general population, rather than specific sub-groups. Particularly as there is no evidence to suggest that people of fair phenotype are clustered in the upper income brackets, as compared to the lowest, these results have no bearing on current programmes.

The sun exposure control programmes which are directed at parents generally are also intended to protect against NMSC. In addition, there are programmes directed at mature adults in Alberta. These study results do not suggest a cogent reason to alter the current interventions in order to maximize the public health benefits.

Targeting specific SES groups for intervention to reduce incidence of lip and testicular cancers is not justified by these results. The group at highest risk of lip cancer consisted of primarily outdoor workers, who can be readily identified through their occupational/industrial classification. Other than general recommendations on smoking cessation and protection from sunlight, the most important requirement for lip cancer is further research to clarify the roles of already suggested risk factors and to uncover any new ones. Although excess risk for testicular cancer was associated with high SES and comparative youth, the recommendation is that education about self-examination be directed at all men below the age of 45 to 50, regardless of SES. The direct determinants of testicular cancer are not sufficiently well known to suggest other interventions.

Unfortunately, this analysis has not contributed substantially to knowledge for the practical development of interventions which could be of assistance in prevention of cancer at many sites. Although one or more of the SES indices were significantly associated with risk for prostatic and brain cancers and with Hodgkin's disease, the fundamental causes of these neoplasms remain unknown and meaningful interventions cannot be proposed.

The final objective of this analysis was to establish whether SES is an indicator of direct determinants of cancer, a potential confounder or an effect modifier. The results suggest that one index, occupational prestige, is a potential confounder. The prestige categories are correlates of job title, which in turn may be associated with hazardous job exposures. It is unfortunate that the prestige categories appear to be imperfect correlates of job exposure, resulting in effects which are difficult to explain. Excess risk in a middle prestige rank, relative to the lowest and highest, for example, is more likely related to job exposures than to the prestige attached to the occupations. On the other hand the occupational prestige classification system may no longer reflect hierarchical positions accurately, if the social status of a number of occupations has changed over time. Income and education were both risk factors for some cancers, presumably as correlates of other, more direct disease determinants, rather than being themselves causal.

There were few significant instances of interaction. Smoking was an effect modifier for lung cancer risk, with inconsistent effects by levels of income and education. Although there were more people with high levels of both who had never smoked, for men who did smoke, the amount was greatest for the high income-education groups. Age interacted with education in malignant melanoma, but the interaction appeared to be a statistical artifact, a result of discordant age distributions among cases relative to controls.

In summary, analysis of the Alberta data suggested that income level would be an appropriate, readily available index for identifying population sub-groups at high risk for lung cancer and for cutaneous malignant melanoma.

Education, although closely associated with cancer risk for some cancer sites, was less effective as an index. Occupational prestige level appeared to be a confounding index because of its inconsistency, both as correlate of job exposure and as a scale of hierarchial position. A combined SES index, using income, education and occupational prestige, cannot be recommended on the basis of this analysis.

A number of deficiencies became apparent during the course of this study, which suggest several areas for future research. When data collection is complete for the Occupational Monitoring project, the data set will be analyzed using as the main variable of interest job titles categorized according to common exposures in the workplace. Income and education will be included as co-variates, together with age, smoking and alcohol to test for effects not detected in the current analysis, which might strengthen the evidence for the use of income or education to identify high risk groups. If numbers permit, it would be valuable to subdivide skin cancers for analysis by histological type, to assess possible differences in determinants for malignant melanoma, squamous cell carcinoma and basal cell carcinoma. Similarly, subdivision of lung cancer cases into adenocarcinomas, small cell and squamous cell may expose differences in risk factors or magnitude of effects.

For future studies using individual indices of SES, it would be interesting to establish family income from all sources, to compare the results with those derived using solely earned income among men. If there is continuing interest in occupational prestige as a variable, the scale should be updated to reflect the attitudes of the Canadian population towards the status of occupations in the 1990s. Women should be included in future studies, in case the results from studying males do not apply to women.

The effectiveness of already existing intervention programmes should be evaluated, in terms of the requirements of population sub-groups most at risk. The degree of behaviour change should be assessed among these groups, compared to similar groups not targeted for intervention. Better knowledge is required about the distribution of the basic determinants of cancer and their relations with various SES indices. For cancer sites where fundamental causes are unknown future exploration of risk factors should include controlling for income level in analysis or through case-control matched selection. Removal of income as a covariable may improve the probability of detecting new associations or confirming effects already detected, but with weak or questionable associations only.

About 11% of all cancers in Alberta in 1990 consisted of lung cancer. Low income was closely associated with high risk in this study. Although income is unlikely to be a direct determinant of lung cancer, the causal role of smoking is beyond doubt. Cigarette smoking is also responsible for about 25% of all deaths among Canadians aged 35-84.[162] The public health benefits of reduction of smoking are thus obvious. It is also clear that smoking cessation is least common among people of low income and education, despite the application of a variety of interventions.[163-166] It has been suggested that smoking may be a coping mechanism through which disadvantaged people deal with the stress associated with living on a low income.[167] Regardless of the cause, it seems clear that efforts to raise the incomes and educational levels of low earners should be emphasized as a measure to promote public health. It will also be crucially important in Canada's present recessionary economic climate to enure that the number of low income earners and of poorly educated people does not increase, as the current situation seems to suggest might happen. The public health impact of such an increase might create an economic burden which society could ill afford.

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APPENDIX 1

AUTHOR'S INVOLVEMENT WITH PROJECT

PERSONAL INVOLVEMENT

The author of this manuscript has been involved with the Occupational Monitoring project since its planning stages, prior to 1983. The author assisted with developing the protocol, pilot-tested the questionnaire (adapted from previous work done in occupational cancer by the author), and did data analysis for the first annual report. In 1985 she was appointed a principal investigator and became responsible for the project. Currently, her duties include supervising staff who are involved in the project (research assistants, data entry clerk, programmer analyst), planning and supervising data analysis, interpreting and reporting the results. She is also responsible for responding to requests for additional data relating to the workplace and possible cancer risks from Alberta Occupational Health and Safety and from Alberta Workers' Compensation Board . The author also participates in the yearly negotiations and application for continued funding.

APPENDIX II

OCCUPATIONAL PRESTIGE RANKS

01 Self-Employed Professionals

Architects
Lawyers, Notaries
Physicians, Surgeons
Dentists
Veterinarians

02 Employed Professionals

Accountants, Auditors, Financial Officers
Chemists
Geologists
Physicists
Meteorologists
Agriculturalists
Biologists, Related Scientists
Engineers
Surveyors
Mathematicians, Statisticians, Actuaries
Economists
Sociologists, Anthropologists, Related Scientists
Psychologists
Social Workers
Judges, Magistrates
Library, Museum, Archival Sciences Supervisors, Librarians, Conservators
Educational, Vocational Counsellors
Ministers of Religion
University Teachers
Kindergarten, Elementary, Secondary, Post-Secondary Teachers
Teachers of Exceptional Studies
Pharmacists
Dieticians, Nutritionists
Commissioned Officers, Armed Forces

03 High Level Management

Members of Legislative Bodies
Government Administrators
General Managers
Management Occupations in Natural Sciences, Engineering, Social Science, Teaching, Medicine, Health, Finance, Sales, Advertising, Transport, Communication

04 Semi Professionals

Drafting Occupations
Systems Analysts, Computer Programmers

Occupations in Welfare, Community Services
Nuns, Brothers, Other Religionists
Community College, Vocational Teachers
Fine Arts Teachers
Instructors, Training Officers
Osteopaths, Chiropractors
Nursing, Therapy Supervisors
Registered, Graduate Nurses
Physiotherapists
Optometrists
Dispensing Opticians
Occupations in Fine Art, Commercial Art, Photography, Performing Arts, Audiovisual Arts, Writing
Supervisors, Arts, Recreation
Athletes
Air Pilots, Navigators, Flight Engineers
Deck Officers

05 Technicians

Physical Science, Life Science, Architectural, Engineering, Library, Museum, Archival Radiological, Medical Laboratory Technologists and Technicians
Denturists
Inspecting, Testing Grading, Sampling Occupations in Mineral Ore Treating, Metal Processing, Chemical, Petroleum, Rubber, Plastic Materials Processing, Metal Shaping, Forming, Fabricating, Assembling Metal Products, Electronic, Electric Power, Lighting, Wire, Communications, Construction, Farming, Horticulture, Animal Husbandry
Respiratory Technicians
Dental Hygienists, Assistants, Laboratory Technicians
Air Transport Operating Support Occupations
Ship's Engineering Officers

06 Middle Management

N.E.C. Administrators, Officials Unique to Government
Management of Personnel, Industrial Relations, Purchasing, Services, Production, Construction, Transport
Personnel Officers
Purchasing Officers, Buyers
Coaches, Trainers, Instructors, Referees in Sports, Recreation
Funeral Directors, Embalmers

07 Supervisors

White Collar Supervisors
Government Inspectors, Regulators

08 Foremen

Captains, Other Officers, Fishing Vessels
Blue Collar Foremen

09 Skilled Clerical/Sales/Service

Library, Museum, Archival Clerks
Nursing Assistants
Radio, Television Announcers
Occupations in Performing, Audiovisual Arts
Secretaries, Stenographers
Bookkeepers, Accounting Clerks
Data Processing Equipment Operators
Claim Adjusters
Technical Salesmen
Commercial Travellers
Insurance, Securities, Real Estate, Advertising, Business Services Salesmen and Agents
Telegraph Operators
Motion-Picture Projectionists

10 Skilled Crafts and Trades

Police, Private Investigators, Fire Fighters
Forestry Conservation
Log Inspecting, Grading, Sealing, Hoisting, Sorting, Moving
Metal Processing Occupations
Clay, Glass, Stone Processing Occupations
Chemical, Petroleum, rubber, Plastic Processing Occupations
Sawyers
Tool and Die Making
Metal Machining Inspecting, Testing
Sheet Metal Workers
Welding, Flame Cutting
Boilermakers, Platers, Structural Metal Workers
Engravers, Etchers
Business, Commercial Machine, Metal Products Fabricating, Assembly, Repair
Repair, Radio, Television, Electrical Equipment
Cabinet, Wood Furniture Makers
Furriers
Mechanics, Repairmen
Electric Power Linemen
Construction Electricians, Repairmen
Wire Communications Equipment Installing, Repair
Pipefitting, Plumbing, Structural Metal Erectors, Glaziers
Air Transport Operating Occupations NEC

Locomotive Engineers, Firemen
Printing, Photoengraving
Power Station Operators
Stationary Engine, Utilities Equipment Operating
Broadcasting Equipment Operating
Audiovisual Recording, Reproduction Equipment Operating
Communications Equipment Operating

11 Farmers

Farm Owners, Operators, Managers

12 Semi Skilled Clerical/Sales/Service

Nursing Aids, Orderlies
Typists, Clerk Typists
Tellers, Cashiers
Bookkeeping, Account Recording Clerks
Office Machine Operator
Material Scheduling, Recording, Distributing Clerks
Reception, Information, Mail Clerks
Sales Clerks
Bartenders, Waitresses, Stewards
Barbers, Hairdressers
Guides
Hostesses

13 Semi Skilled Crafts/Trades

Other Ranks, Armed Forces
Chefs, Cooks
Fishermen
Timber Cutting
Rotary Well, Rock, Soil Drilling
Blasting, Mining, Quarrying
Mineral Ore Treating Occupations
Food, Beverage Processing
Wood, Pulp, Paper Processing
Textile, Tobacco, Hide, Pelt Processing
Machine Tool Operating
Forging
Wood, Clay, Stone, Glass Machining
Fabricating, Assembling Aircraft, Mechanized Equipment, Machinery, Precision Instruments
Assembly Repair Electrical Equipment, Wood Products, Textiles, Fur, Leather, Rubber, Plastic
Mechanics, Repairmen NEC
Excavating, Grading, Paving, Concrete Finishing, Insulating, Roofing, Waterproofing

Transport Support Occupations
Photographic Processing

14 Unskilled Clerical

Mail, Message Distribution Occupations
Hotel, General Office Clerks
Commodities Sales NEC
Street Vendors
Door to Door Salesmen
NEC Occupations In Food, Beverage Preparation, Lodging, Accommodation, Personal Service
Babysitters

15 Unskilled Labourers

Attendants and NEC Occupations, Sports, Recreation
Security Guards
Cleaners
Porters
Laundering, Dry Cleaning, Pressing
Janitors
Elevator Operators
Trapping, Fishing
Labourer, Forestry, Logging, Mining, Quarrying, Oil, Gas, Mineral Ore Treating, Metal Processing, Clay, Glass, Stone Processing, Chemicals, Petroleum, Rubber, Plastics Processing
Food Preserving Occupations
Labourer, Wood, Pulp, Papermaking, Textiles
Wood Machining
Labourer, Fabricating, Assembly, Various Products
Railway Section, Track Workers
Labourers, Excavating, Grading, Paving
Labourers, Electric Power, Lighting, Wire Communications Installation, Repair
Construction, Transport Labourers

16 Farm Labourers

Livestock, Crop, Nursery Workers
Farm Machinery Operators

APPENDIX III

QUESTIONNAIRE

Alberta Cancer Board Environmental Profile Questionnaire

INSTRUCTION SHEET

Hello

The Environmental Profile Questionnaire is part of the Alberta Cancer Board's ongoing efforts to learn more about health risks and the causes of cancer. Please assist us by completing this questionnaire and returning it in the stamped, self-addressed envelope.

We hope that the questionnaire isn't difficult to complete. Where a set of responses is listed (like in a multiple choice exam) simply choose the response that is best for you and print the **number** in the box provided. The response **N/A** means 'not applicable' and use this response if the question does not apply to you. Where you are requested to provide an answer, just **print** it on the **line** (I have emphasized print because if you're anything like me..!). Please do not mark in the shaded areas as they are for use at the office.

If you do have any problems, please call for assistance to our **Research Assistant, Ms. Heather Jordan** at **482-9377** (call collect from outside Edmonton).

Thank you for your time and cooperation.

Please remove this instruction sheet before sending the questionnaire back to us.

Alberta Cancer Board Environmental Profile Questionnaire

BASIC INFORMATION SECTION

A. Today's date:

Day ☐ Month ☐ Year 19☐

B. This questionnaire was completed by:

1 Myself

2 A Relative

3 A Friend ☐

C. Your sex is:

1 Male

2 Female ☐

D. Your current marital status is:

1 Married or common-law
2 Separated or divorced
3 Widowed
4 Single, never married

E. Date of Birth:

Day ☐ Month ☐ Year 19☐

F. In what city or town were you born?

____

G. In what province were you born? (Mention the country if you were born outside of Canada.)

____

H. How many years in total have you lived in Alberta? (Round your response to the nearest whole year.)

☐Years

I. How many years in total have you lived in Canada? (Round you response to the nearest whole year.)

☐Years

J. What is your religious preference?

1 Seventh Day Adventist
2 Jewish
3 Protestant
4 Hutterite
5 Mormon
6 Roman Catholic
7 Other (specify)
8 No Religious Preference

☐

Alberta Cancer Board Environmental Profile Questionnaire

BASIC INFORMATION SECTION

K. Please indicate the number of years you completed at each of the following types of school.

Elementary & Secondary (grades 1-13)

☐Years

University or College

☐Years

Technical, Vocational or Trade School

☐Years

Other (specify)

☐Years

L. What is your current employment status?

1 Employed
2 Self-employed
3 Retired
4 Part-time employed
5 Unemployed
6 Student
7 Other (specify)

☐

Following are a few questions for women only about their reproductive history. Male respondents may continue to the Family History section.

M. How old were you when you had your first menstrual period?

☐Years
-9 Unknown

N. How many times have you been pregnant?

☐Number

O. If applicable, how old were you at menopause (that is, when you stopped menstruating).

☐Years
-8 N/A
-9 Unknown

Thank you. Please continue to the Family History section.

Alberta Cancer Board Environmental Profile Questionnaire

FAMILY HISTORY SECTION

The following questions refer only to immediate family members who are not adopted.

A. How many sisters and half-sisters do (or did) you have?

☐Number

B. How many brothers and half-brothers (or did) you have?

☐Number

C. How many daughters do (or did) you have?

☐Number

D. How many sons do (or did) you have?

☐Number

E. How many members of your immediate family (your mother, father, sisters, brothers, children or spouse) have ever had cancer?

☐Number
-9 Unknown

In the following sections, please provide information about any direct relative who had cancer. Tell us who the relative was and where they had cancer.

F. One relative having cancer was your:
1 Mother
2 Father
3 Sister or Half-Sister
4 Brother or Half-Brother
5 Daughter
6 Son
7 Spouse

☐

G. Where in their body did they have cancer?

____

H. Another relative with cancer was your:

1 Mother
2 Father
3 Sister or Half-Sister
4 Brother or Half-Brother
5 Daughter
6 Son
7 Spouse

☐

I. Where in their body did they have cancer?

____

Alberta Cancer Board Environmental Profile Questionnaire

FAMILY HISTORY SECTION

J. Another relative with cancer was your:

1 Mother
2 Father
3 Sister or Half-Sister
4 Brother or Half-Brother
5 Daughter
6 Son
7 Spouse

☐

K. Where in their body did they have cancer?

____

L. Another relative with cancer was your:

1 Mother
2 Father
3 Sister or Half-Sister
4 Brother or Half-Brother
5 Daughter
6 Son
7 Spouse

☐

M. Where in their body did they have cancer?

____

N. Another relative with cancer was your:

1 Mother
2 Father
3 Sister or Half-Sister
4 Brother or Half-Brother
5 Daughter
6 Son
7 Spouse

☐

O. Where in their body did they have cancer?

____

P. Another relative with cancer was your:

1 Mother
2 Father
3 Sister or Half-Sister
4 Brother or Half-Brother
5 Daughter
6 Son
7 Spouse

☐

Q. Where in their body did they have cancer?

____

Alberta Cancer Board   Environmental Profile Questionnaire

FAMILY HISTORY SECTION

R	Another relative with cancer was your:

1 Mother
2 Father
3 Sister or Half-Sister
4 Brother or Half-Brother
5 Daughter
6 Son
7 Spouse

☐

S.	Where in their body did they have cancer?

____

Thank you. Please proceed to the Tobacco and Alcohol Use section.

Alberta Cancer Board Environmental Profile Questionnaire

TOBACCO AND ALCOHOL USE SECTION

TOBACCO USE

A. Have you ever smoked cigarettes, cigars, a pipe, or used chewing tobacco?

1 No 2 Yes ☐

B. How many people living in your home do (or did) smoke daily (not including yourself)?

☐Number

C. At your job outside your home, are (or were) you exposed to tobacco smoke?

1 Yes, all the time
2 Yes, some of the time
3 No

☐

If you have never smoked, then please proceed to the Alcohol Use questions in the second column on the back of this page.

D. At what age did you start smoking?

☐Years

E. For each product listed below, indicate if you presently use it daily, occasionally or not at all.

Cigarettes ☐
1 Daily 2 Occas'ly 3 Never

Cigars ☐
1 Daily 2 Occas'ly 3 Never

A Pipe ☐
1 Daily 2 Occas'ly 3 Never

Chewing Tobacco or Snuff ☐
1 Daily 2 Occas'ly 3 Never

The following questions apply to cigarette smokers only. *If you have never smoked cigarettes, please continue with the Alcohol Use questions in the second column on the back of this page.*

F. Do you (or did you) normally smoke plain or filtered cigarettes?

1 Plain
2 Filtered
3 Both plain and filtered

☐

Alberta Cancer Board Environmental Profile Questionnaire

TOBACCO AND ALCOHOL USE SECTION

G. How many **cigarettes** do you (or did you) normally smoke per day?

☐Number

H. For how many years have you smoked (or did you smoke) cigarettes at this rate?

☐Years

I. If you have stopped smoking **permanently,** how long has it been?

1 Less than one year
2 One to five years
3 More than five years
-8 N/A

☐Number

Please continue with the Alcohol Use questions.

ALCOHOL USE

J. Have you ever had alcoholic beverages?

1 No 2 Yes ☐

*If you answered **NO** to the question above, proceed to the Residential History section.*

K. At what age did you begin to drink alcoholic beverages?

☐Years

L. How many years have you used (or did you drink) alcoholic beverages?

☐Years

M. For each of the types of beverages shown below please indicate how much you usually drink each month (or how much you usually drank each month in the past).

Beer (bottles or cans) ☐Number

Wine (4-5 oz. glasses) ☐Number

Spirits (1.5 oz of liquor) ☐Number

Thank you. Please continue to the Residence History section.

Alberta Cancer Board Environmental Profile Questionnaire

RESIDENTIAL HISTORY SECTION

PLACES WHERE YOU HAVE LIVED

Please list all the places where you have lived at least one year or more. Begin with your present residence (no matter how long you have lived there) and work backwards to your childhood homes. Do not include moves within one city or town. For each location, please indicate if this was a "farm" home or "other".

EXAMPLE

	Place Name (City, Town or District)	Location (Province if Canada Otherwise Country)	Years From	Years To	Was this a Farm Home?
1	____________	____________	19 ☐	19 ☐	1 Farm 2 Other

	Place Name (City, Town or District)	Location (Province if Canada Otherwise Country)	Years From	Years To	Was this a Farm Home?
1	____________	____________	19 ☐	19 ☐	1 Farm 2 Other
2	____________	____________	19 ☐	19 ☐	1 Farm 2 Other
3	____________	____________	19 ☐	19 ☐	1 Farm 2 Other
3	____________	____________	19 ☐	19 ☐	1 Farm 2 Other
5	____________	____________	19 ☐	19 ☐	1 Farm 2 Other
6	____________	____________	19 ☐	19 ☐	1 Farm 2 Other
7	____________	____________	19 ☐	19 ☐	1 Farm 2 Other

There is space for more residential history information on the back of this page.

Environmental Profile Questionnaire   Residential History Section

8____________________ ____________________ 19 ☐ 19 ☐ 1 Farm 2 Other

9____________________ ____________________ 19 ☐ 19 ☐ 1 Farm 2 Other

10____________________ ____________________ 19 ☐ 19 ☐ 1 Farm 2 Other

11____________________ ____________________ 19 ☐ 19 ☐ 1 Farm 2 Other

Thank you. Please continue to the Employment History section.

Alberta Cancer Board Environmental Profile Questionnaire

EMPLOYMENT HISTORY SECTION

PLACES WHERE YOU HAVE WORKED

Please list all the places where you have worked at least one year or more. Begin with your present or most recent employment and work backwards to your first job. Include any years that were spent at home. Please be as specific as possible when you describe your activities. For example, indicate if you were a livestock farmer or a grain farmer; a bank clerk or a sales clerk.

EXAMPLE

Job Title	Type of Business or Industry	Place Name (City, Town) or District	Location (Province or Country)	Years From	Years To
1				19 ☐	19 ☐
2				19 ☐	19 ☐

Job Title	Type of Business or Industry	Place Name (City, Town) or District	Location (Province or Country)	Years From	Years To
1				19 ☐	19 ☐
2				19 ☐	19 ☐
3				19 ☐	19 ☐
4				19 ☐	19 ☐
5				19 ☐	19 ☐
6				19 ☐	19 ☐
7				19 ☐	19 ☐

Environmental Profile Questionnaire Employment History Section

8 ________ ________ ________ ________ 19 ☐ 19 ☐

9 ________ ________ ________ ________ 19 ☐ 19 ☐

10 ________ ________ ________ ________ 19 ☐ 19 ☐

11 ________ ________ ________ ________ 19 ☐ 19 ☐

Thank you. Please proceed to the Exposure History section.

Alberta Cancer Board Environmental Profile Questionnaire

EXPOSURE HISTORY SECTION

EXPOSURES

Sometimes people work or live in places where they breathe **DUST, SMOKE, FUMES** or **SPRAYS.** Occasionally their **SKIN** (eg. hands) or **CLOTHING** is exposed to these materials. If you have been exposed for **at least one year or more** to any of the following materials, indicate accordingly. If you were exposed in your job, hobby or home, please **circle** the appropriate response. Also state the years you were exposed, how many months of the year and how many days of the month you were exposed. If you have been exposed to any noxious or toxic substance that is not included in this list, please add it in the space provided for 'Others' on the last page of this section.

EXAMPLE

Material	**Where were you exposed?**	**Year From**	**Year To**	**Months per year**	**Days per month**
Uranium (67)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐

A. DUST

Material	**Where were you exposed?**	**Year From**	**Year To**	**Months per year**	**Days per month**
Asbestos (10)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Cement (11)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Coke (coal fuel) (12)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐

Environmental Profile Questionnaire Exposure History Section

A. DUST
Cont.

Material	**Where were you exposed?**	**Year From**	**Year To**	**Months per year**	**Days per month**
Fiberglass (13)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Nickel (14)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Leather (15)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Wood (16)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Stone (17)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Coal (18)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Grain (19)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Cotton, Canvas (21)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Silica (23)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐

Environmental Profile Questionnaire Exposure History Section

B. CHEMICALS

Material	Where were you exposed?	Year From	Year To	Months per year	Days per month
Fertilizers (30)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Diesel fuel (31)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Coal tar (32)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Ink (33)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Paints (34)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Dyes (35)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Herbicide (36)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Fungicide (37)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐

Environmental Profile Questionnaire Exposure History Section

B. CHEMICALS **cont.** **Material**	**Where were you exposed?**	**Year From**	**Year To**	**Months per year**	**Days per month**
Insecticide (38)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Cutting oils (39)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Crude petroleum (40)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Solvents (41)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Cleaning fluids (42)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Ether (43)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Printing fluids (44)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Benzene (45)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐

Environmental Profile Questionnaire Exposure History Section

B. CHEMICALS

Cont. Material	Where were you exposed?	Year From	Year To	Months per year	Days per month
PVC (46)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Naphthylamines (47)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Hydrazine (48)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Formaldehyde (49)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐

C. METALS

Arsenic (55)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Chromium (56)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Nickel (57)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Iron ore (58)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐

Environmental Profile Questionnaire Exposure History Section

B. METALS **Cont.** **Material**	**Where were you exposed?**	**Year From**	**Year To**	**Months per year**	**Days per month**
Cadmium (59)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Lead (60)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Beryllium (63)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
D. RADIATION					
Work with X-rays (65)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Radium (66)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Uranium (67)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Nuclear isotopes (68)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
E. LIGHTING					
Ultraviolet light (70) (sunlight)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐

Environmental Profile Questionnaire Exposure History Section

D. LIGHTING Cont. Material	**Where were you exposed?**	**Year From**	**Year To**	**Months per year**	**Days per month**
Fluorescent lighting (71)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Video display terminals (72)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
F. FUMES					
Wood smoke (74)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Exhaust fumes (75)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Coke oven emissions (76)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Natural gas (77)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Sour gas (H_2S) (78)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Welding fumes (79)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐

Environmental Profile Questionnaire Exposure History Section

G. PROCESSES

Cont. Material	**Where were you exposed?**	**Year From**	**Year To**	**Months per year**	**Days per month**
Plastics manufacturing (81)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Rubber Manufacturing (82)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Battery manufacturing (83)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Tar sands processing (84)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Petrochemical processing (85)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Furniture manufacturing (86)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
Aluminum processing (87)	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐

Environmental Profile Questionnaire   Exposure History Section

	Where were you exposed?	Year From	Year To	Months per year	Days per month
OTHERS? **(please specify)**					
	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐
	1 Job 2 Hobby 3 Home	19 ☐	19 ☐	☐	☐

THIS IS THE END OF THE QUESTIONNAIRE

Would you be willing to take part in other projects of this nature?

1 No 2 Yes ☐

Thank you very much for your cooperation. Please feel free to add any further comments you think may be helpful.

APPENDIX IV

TOPOGRAPHY AND MORPHOLOGY OF CANCER SITES

Site Title[10]		*ICDO-0 Codes*[11]	*ICDO-9 Codes*
1.	Lip (External Lip, Mucosa, Commissure, Excludes Skin)	C00	140
2.	Other Head/Neck (Tongue, Tonsils, Mouth Excluding Lip, ?/Salivary Glands, Oropharynx, Hypopharynx	C01.9, C02 to C10, C13, C14	141-146, 148
3.	Nasopharyngeal/Sinonasal (Nasopharynx, Sinuses, Nasal Cavity, Inner Ear	C11, C12.9, C30.0, C31	147, 160.0, 160.2
4.	Oesophagus	C15	150
5.	Stomach	C16	151
6.	Colon/Rectosigmoid Function	C18, C19	153, 154.0
7.	Rectum	C20	154.1-154.8
8.	Liver	C22.0	155.0
9.	Gallbladder/Intra-/Extrahepatic Bile Ducts	C23.9, C24, C22.1	156, 155.1
10.	Pancreas	C25	157
11.	Larynx	C32	161
12.	Lung/Trachea/Bronchus	C33.9, C34	162
13.	Bone/Connective Tissue (Bones, Joints, Articular Cartilage, Connective, Subcutaneous, Other Soft Tissues)	C40 to C41, C49	170, 171
14.	Malignant Cutaneous Melanoma	C44+ M872 to 879	172
15.	Non-melanotic Skin (Excluding Skin of Penis, Scrotum and Malignant Cutaneous Melanoma)	C44 (excluding M872-M879)	173
16.	Male Breast (excluding Skin)	C50	175

---

[10] All site groups include Not Otherwise specified (NOS).

[11] All morphologies are malignant (behaviour code 3).

Site Title[12]		*ICDO-0 Codes*[13]	*ICDO-9 Codes*
17.	Prostate	C61.9	185
18.	Testis	C62	186
19.	Bladder	C67	188
20.	Kidney/Renal Pelvis/Ureter/Urethra	C64.9, C65.9, C66.9, C68	189
21.	Brain/Central Nervous System (Meninzes, Cerebrum, Cerebellum, All Labea, Ventricle, Brain Stem, Spinal Cord, Olfactory/Optic/Acoustic/Cranial/ Peripheral Nerves, Autonomic Nervous System)	C70, C71, C72, C47 (all tumours)	191, 192
22.	Non-Hodgkin's Lymphoma/Lymph Nodes (Grandular Cell Tumour, Alveolar Soft Part Sarcoma, Lymphosarcoma, Reticulosarcoma, Cutaneous, Peripheral T-Cell)	C77, M958, M959, M967 to M972	200, 202
23.	Hodgkin's Disease/Lymph Nodes	C77, M965 to M966	201
24.	Multiple Myeloma/Bone Marrow (Plasmocytoma, Multiple Myeloma)	C42.1, M9731 to 9732	203
25.	Leukemia/Bone Marrow (Acute, Chronic, Aleukemic, Myelocytic, Monocytic, Hairy Cell, Mast Cell)	C42.1, M980 to 994	205-208
26.	Thyroid Gland	C73.9	193

---

[12] All site groups include Not Otherwise specified (NOS).

[13] All morphologies are malignant (behaviour code 3).

APPENDIX V

SPECIFICATION OF THE MODEL

Multiple Logistic Regression Model for Estimating ORs

1. Model: In general, the model can be specified as

$$\log\frac{p}{1-p}=\alpha+B_{11}D_{11}+B_{12}D_{12}+\ldots+B_{1k_1}D_{1k_1}+B_{21}D_{21}+\ldots$$

where p = probability of disease

and D_{1i} , $i=1,2,\ldots k$, are dummy variables associated

with a covariate V_1 with $K_1 + 1$ categories, of which one category is

chosen as baseline for comparison. Thus if V_1 takes the baseline value,

$D_{1i} = 0$, for all i

If V_1 takes the value of the first category, $D_{11} = 1$ and $D_{1i} = 0$ for $i \neq 1,\ldots$.

The model is estimated using backward selection and B_{1i}

facilitates comparison of disease odds between the i^{th} category and the baseline.

2. Example: Lip Cancer

Covariate	# Categories	# Dummy Variables
age	2	1 (agegp1)
income	3	2 (I1_hi, I1_mid)
education	3	2 (E1_hi, E1_mid)
COP	2	1 (cop1)

3. Variable Levels

Agegp1 = ≤50 years Baseline = >50 years

I_1-hi = ≥$20,000, I_1_mid = $10,000-19,999 Baseline = <$10,000

E_1_hi = university education, E_1_mid = secondary or technical education
Reference = elementary education

Cop1 = $\text{Cop}_{\text{I-IV}}$ Baseline = $\text{Cop}_{\text{V+VI}}$ (Farm Owners + Farm Labourers + Blue Collar Other).

APPENDIX VI

CURRICULUM VITAE

SHIRLEY M. FINCHAM

Curriculum Vitae

PRESENT ADDRESS: 11011 - 80 Avenue, Edmonton, Alberta T6G 0R2

DEGREES:

B.A., Dept. of English
University of Alberta, 1955

B.A. (Hon), Dept. of Psychology
University of Saskatchewan, 1969

M.A., Dept. of Psychology
University of Saskatchewan, 1972

Ph.D. (candidate), Dept. of Sociology
University of Alberta, 1995

AWARDS:

University Graduate Scholarship
University of Saskatchewan, 1970

POSITIONS HELD:

Past:

Research Assistant 1970-72
Department of Psychology
University of Saskatchewan
Saskatoon, Saskatchewan

Research Assistant 1974-81
Department of Biostatistics,
Analysis and Cancer Registry
Cross Cancer Institute
Provincial Cancer Hospitals Board
Edmonton, Alberta

Acting Director January 1-June 1989
Division of Epidemiology
and Preventive Oncology
Alberta Cancer Board
Edmonton, Alberta

Current:

Research Scientist 1981-present
Division of Epidemiology,
Prevention and Screening
Alberta Cancer Board
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Adjunct Professor 1987-present
Department of Public Health Sciences
Faculty of Medicine, University of Alberta
Edmonton, Alberta

PUBLICATIONS:

1. **SM Fincham** and JA Mills. Implicit phonological rules in children: an examination of Messer's and Menyuk's work. The Journal of Psychology 88: 175-189, 1974.
2. M Grace, WC Taylor, EN Skakun, and **S Fincham**. Computerized patient management problems: an alternative examination technique. Proceedings Fourteenth Annual Conference on Research in Medical Education, Association of American Medical Colleges, Washington, D.C., 1975.
3. WC Taylor, M Grace, TR Taylor, **SM Fincham** and EN Skakun. The use of computerized patient management problems in a certifying examination. Medical Education 10:404-407, 1976.
4. **SM Fincham**, M Grace, WC Taylor, EN Skakun and FG Davis. Paediatric candidates' attitudes to computerized patient management problems in a certifying examination. Medical Education 10:404-407, 1976.
5. M Grace, J Hanson, **SM Fincham**, EN Skakun and WC Taylor. A scoring technique for computerized patient management problems. Medical Education 11:335-340, 1977.
6. EN Skakun, WC Taylor, M Grace and **S Fincham**. A preliminary investigation of CPMPs in relation to other examinations. Ed. Psych. Meas. 39:1979.
7. RD Egedahl, **SM Fincham**. Annotated Bibliography on Occupational Disease in Alberta. Provincial Cancer Hospitals Board, Edmonton, 1977.
8. **SM Fincham**, RD Egedahl, M Grace, W Wark, J Hanson and R Dewar. Occupational Malignancies Among Petrochemical Workers in Alberta. Provincial Cancer Hospitals Board, Edmonton, 1979.
9. **S Fincham** and GB Hill. Mesothelioma of the pleura, peritoneum and tunica vaginalis testis - a review of 33 cases. Chronic Diseases in Canada (4):73-77, 1983.
10. GB Hill, **S Fincham**, C Wijayasinghe, C Haronga and M Hendin. Sex ratio of offspring of patients with prostatic cancer. Can Med Assoc J September 1985:Vol. 133;567-571.
11. B Alleyne, S Campbell, **S Fincham**, GB Hill, J Kalnas. Studies of occupational cancer using mortality records and cancer registrations. Alberta Occupational Medicine Newsletter, Spring 1986, Vol.IV, No. 1:5-7.
12. RP Gallagher, JM Elwood, WJ Threlfall, JJ Spinelli, **SM Fincham**, GB Hill. Socio-economic status, sunlight exposure and risk of malignant melanoma: The Western Canada Melanoma Study. J Natl Cancer Inst Vol. 79, No. 4, October 1987.
13. **S Fincham**, J Kalnas, GB Hill, R Meleshko, B Alleyne, C Wijayasinghe. Monitoring of occupations in Alberta cancer patients. In: G.W. Gibbs, J. Markham eds. Proceedings of a Conference Organized by the Canadian Occupational Health Association. Occupational Health Services in Canada Through the Year 2000. 1988;214-220.

14. GB Hill, **SM Fincham**, HH McDuffie, T To, JA Dosman. Relationship between pesticide use and the incidence of soft tissue sarcoma, Hodgkin's disease, non-Hodgkin's lymphoma and multiple myeloma. Chronic Diseases in Canada 1988; Vol 9, No. 6(Nov):113-116.

15. **S Fincham**, GB Hill, J Hanson, C Wijayasinghe. The epidemiology of prostatic cancer - a case-control study. Prostate 17(3):189-206, 1990.

16. SE Hrudey, CL Soskolne, J Berkel, **S Fincham**. Drinking water fluoridation and osteosarcoma. Can J of Public Health 81(6):415-416, 1990.

17. H Jordan, J Berkel, **S Fincham**. Random Digit Dialling for Control Selection. Chronic Diseases in Canada 1991; Vol. 11, No. 2(Mar).

18. **S Fincham**, J Hanson, J Berkel. Cancer patterns and risks in Alberta farmers. Cancer 1992;69(5):1276-1285.

19. **S Fincham**. Community Health Promotion Programs. Social Sci Med 1992; 35:239-249.

20. **S Fincham**, A MacMillan, D Turner, J Berkel. Occupational risks for cancer in Alberta. Health Rep 1993;5(1)67-72.

21. **S Fincham**, A MacMillan, J Berkel. Cancer patterns among female farmers in Alberta.
    and
22. S Gabos, **S Fincham**, J Berkel. The interaction between farming occupation and exposure to smoking in squamous carcinomas of the lip. In: Human Sustainability In Agriculture: Health, Safety, Environment. HH McDuffie, et al (eds). Lewis Publishers, Chelsea, Michigan. In press.

23. R Gallagher, GB Hill, CD Bajdik, **S Fincham**, AJ Coldman, DI McLean, WJ Threlfall. Sunlight exposure, pigmentary factors, and risk of nonmelanocytic skin cancer. Arch Dermatol 1995;131:157-163.

PRESENTATIONS:

"Occupational Carcinogenesis". WF Hall, R Hosein, **SM Fincham** and R Egedahl. Cross Service Rounds, Cross Cancer Institute, Edmonton, November 1977.

"Occupational Cancer Risks in Alberta". Seminar for Canadian Cancer Society Nurses, Cross Cancer Institute, Edmonton, March 1979.

"Occupational Carcinogenesis". Department of Occupational Health Nursing, Grant McEwan College, Edmonton, April 1979.

"Occupational Cancers - Current Topics". Seminar for Canadian Cancer Society Nurses, Cross Cancer Institute, Edmonton, December 1980.

"Risk Factors in Cancer". Seminar for Oncology Nurses, Cross Cancer Institute, Edmonton, January 1981.

"Occupational Monitoring in Alberta - Method, Cost, Problems & Results". Cancer Surveillance Workshop, Ottawa, December 1981.

"Ascertainment of Occupation in Men with Cancer". Research Meeting, Department of Epidemiology, Alberta Cancer Board, Edmonton, May 1981.

"Preliminary Report on Prostate Cancer in Alberta". Research Meeting, Department of Epidemiology, Alberta Cancer Board, Edmonton, December 1981.

"The Alberta Cancer Registry: Identifying Occupational Malignancies". Science Advisory Committee to the Environment Council, University of Calgary, Calgary, March 1982.

"Ischaemic Heart Disease". Community Medicine 411 Seminar, University of Alberta, Edmonton, September 1983.

"Summary for Physician Information - Early Detection of Colorectal Cancer in Alberta". Baker Clinic, Edmonton, October 1982.

"Epidemiology of Bladder Cancer in Alberta". Research Meeting, Department of Epidemiology, Alberta Cancer Board, Edmonton, November 1983.

"Sex Ratio of Offspring of Prostatic Cancer Patients". GB Hill, **S Fincham**, C Wijayasinghe, S Haronga and M Hendin. University of Calgary Rounds, Calgary, February 1985.

"Sex Ratio of Offspring of Prostatic Cancer Patients". GB Hill, **S Fincham**, C Wijayasinghe, S Haronga and M Hendin. Division of Epidemiology, Biometry and Occupational Oncology, British Columbia Cancer Control Agency, Vancouver, March 1985.

"Sex Ratio of Offspring of Prostatic Cancer Patients". GB Hill, **S Fincham**, C Wijayasinghe, S Haronga and M Hendin. American Society of Preventive Oncology (ASPO), Toronto, April 1985.

"A Case-Control Study of Carcinoma of the Skin in Alberta Males". **S Fincham**. Epidemiology Meeting, Clinical Sciences HSA Conference Room, University of Alberta, Edmonton, November 1986.

"Monitoring of Occupations In Alberta Cancer Patients". **S Fincham**, J Kalnas, GB Hill, R Meleshko, B Alleyne, C Wijayasinghe. Occupational Health Services in Canada Through the Year 2000, University of Calgary, Calgary, November 1986.

"Case Control Study of Prostatic Cancer". **S Fincham**, GB Hill, C Wijayasinghe. Annual Meeting of the Royal College of Physicians and Surgeons of Canada and The Canadian Society for Clinical Investigation, Epidemiology Section, Toronto, September 1986.

"Epidemiology of Prostatic Carcinoma". GB Hill, **SM Fincham**, C Wijayasinghe. Presented to Carcinoma of Prostate: Current Approaches, University Hospital, London, Ontario, September 1986.

"Soft Tissue Sarcoma and Pesticides - Current State of Knowledge". **S Fincham**. Ellis Hall, University of Saskatchewan, Saskatoon, September 1987.

"Cancer Patterns in Alberta Farmers". **S Fincham**. Workshop on Cancer in Rural Areas, University of Saskatchewan, Saskatoon, 1989.

"Risk Factors for Non-Melanotic Skin Cancer In Alberta Males". **S Fincham**, GB Hill. Poster presented to the Society for Epidemiological Research Meeting, Birmingham, Alabama, 1989.

"Occupational Diseases In Alberta Workers Exposed to Chemicals - Cancer Incidence". **S Fincham**, J Hanson, GB Hill, B Alleyne. Poster presented to the Alberta Cancer Board Scientific Meeting, Kananaskis, November 1989.

"Risk Factors for Non-Melanotic Skin Cancer in Alberta Males". **S Fincham**, GB Hill. Poster presented to the Alberta Cancer Board Scientific Meeting, Kananaskis, November 1989.

"Cancer Patterns in Alberta Farmers". **S Fincham**, J Hanson. Poster presented to the XIIth Scientific Meeting of the International Epidemiological Association, Los Angeles, California, 1990.

"Cancer Patterns in Alberta Farmers, 1983 - 1988". **S Fincham**, J Hanson. Poster presented to the 15th International Cancer Congress, Hamburg, Federal Republic of Germany, 1990.

"Occupational Risk of Cancer In Alberta". **S Fincham**, J Hanson, J Berkel. Alberta Cancer Board Research Retreat, Banff, November 1990.

"Cancer Patterns and Risks In Alberta Farmers". **S Fincham**, J Hanson, J Berkel. Alberta Cancer Board Research Retreat, Banff, November 1990.

"Occupational Risks for Cancer In Women?". **S Fincham**, J Hanson. Cross Cancer Institute Grand Oncology Rounds, Edmonton, April 1991.

"Cancer Patterns in Alberta Farmers". **S Fincham**, J Hanson, J Berkel. World Environment Conference, Calgary, April 1991.

"Cancer Patterns in Alberta Farmers". **S Fincham**, J Hanson, J Berkel. Poster presented to the 2nd Canadian Epidemiology Research Conference, Edmonton, May 1991.

"Cancer Patterns in Alberta Farmers". **S Fincham**, J Hanson, J Berkel. Poster accepted (but not presented) at The 10th Asia Pacific Cancer Conference, Beijing, August 1991.

"Occupations and Risk Factors Associated with Cancer in Alberta". **S Fincham**, J Hanson, J Berkel. Poster presented to the 2nd Canadian Epidemiology Research Conference, Edmonton, May 1991.

"Monitoring Risks of Cancer in Alberta". **S Fincham**, J Hanson, J Berkel. Third Conference of the International Society for Environmental Epidemiology, Jerusalem, August 1991.

"Radiation as a Risk Factor in Thyroid Cancer". The Canadian Cancer Registries Epidemiological Research Group: S Buehler, N Choi, **S Fincham**, G Hill, N Kreiger, L Marrett, M McBride, D Robson, G Theriault, D Thomson, J Wallace. Alberta Cancer Board Research Retreat, Kananaskis, November 1991.

"Sun Related Risk Factors in Non-Melanoma Skin Cancer". R Gallagher, **S Fincham**, G Hill, CP Yang. 2nd Annual International Symposium, Epidemiology of Malignant Melanoma, Vancouver, October 1991.

"Thyroid Cancer and Radiation". S Buehler, N Choi, **S Fincham**, G Hill, N Kreiger, L Marrett, M McBride, G Theriault, D Thomson, J Wallace. Poster presented to the Society for Epidemiological Research, Buffalo, June 1991.

"Cross Canada Study of Pesticides and Health". JA Dosman, HH McDuffie, D Robson, P Pahwa, **S Fincham**, J McLaughlin. Surgeon General's Conference: Agricultural Safety and Health, Atlanta, 1991.

"Cancer Patterns in Alberta Farmers". **S Fincham**, J Berkel, J Hanson. South East Asia Regional Scientific Meeting of the International Epidemiological Association, Bali, Indonesia, January 1992.

"Cancer Patterns Among Female Farmers in Alberta". **S Fincham**, A MacMillan, J Berkel. Third International Symposium: Issues in Health, Safety and Agriculture, Saskatoon, May 1992.

"Cancer Patterns in Alberta Farmers". **S Fincham**, J Berkel, J Hanson. Third International Symposium: Issues in Health, Safety and Agriculture, Saskatoon, May 1992.

"The interaction between farming occupation and exposure to smoking in squamous carcinomas of the lip". S Gabos, **S Fincham**, J Berkel. Third International Symposium: Issues in Health, Safety and Agriculture, Saskatoon, May 1992.

"Cancer Patterns Among Female Farmers in Alberta". **S Fincham**, A MacMillan, J Berkel. Meeting of the Society for Epidemiological Research, Minneapolis, June 1992.

"Occupational Risks for Cancer in Alberta". **S Fincham**, A MacMillan, D Turner, J Berkel. International Association of Cancer Registries Annual Meeting, Ottawa, June 1992.

"Occupational Surveillance of Alberta Cancer Patients". **S Fincham**, A MacMillan, J Berkel. Summer Scientific Meeting (Joint IEA/ADELPH/Soc Sci Med), Sussex, UK, July 1992. (accepted but not presented)

"Electromagnetic Field Exposure and Cancer Risk in Alberta". **S Fincham**, V Siaw, A MacMillan, J Berkel. Cross Cancer Institute Rounds, Edmonton, 1992.

"Cancer in the Alberta Commercial Printing and Publishing Industry". **S Fincham**, A MacMillan, J Berkel, V Siaw. Canadian Society for Epidemiology and Biostatistics Conference, Quebec City, June 1993.

"Are Policemen at Excess Risk of EMF-related Cancers?". **S Fincham**, A MacMillan, J Berkel, V Siaw. and
"Cancer Risk in Motor Transport Occupations in Alberta". **S Fincham**, A MacMillan, J Berkel, V Siaw. 24th International Conference on Occupational Health, Nice, France, October 1993.

"Cancer Risks In Medicine and Health In Alberta". S **Fincham**, A MacMillan, V Siaw. Accepted but not presented. ISEE/ISEA Joint Conference, Chapel Hill, N.C., March 1994.

"Cancer Risk In Motor Transport Occupations In Alberta". **S Fincham**, A MacMillan, V Siaw, J Berkel Accepted but no presented. Tenth International Symposium, Epidemiology In Occupational Health, Como, Ital, September 1994.

"Occupational Risk for Cancer Among Women". **S Fincham**, A MacMillan, V Siaw. Accepted but not presented. Annual Meeting of the International Association of Cancer Registries, Bangalore, India, October 1994.

"Cancer Patterns In Alberta's Petroleum Industry. **S Fincham**, A MacMillan, D Turner. Accepted but not presented. XVI International Cancer Congress, New Delhi, India, November 1994.

"Risk Factors for Non-Hodgkins' Lymphoma". **S Fincham**, NW Choi, D Robson, J Spinelli, LF Skinnider, D White, HH McDuffie, JM Laughlin, JA Donman, G Theriault, P Pahwa, PZ Wang. Genetics Society of Canada/Canadian Society for Plant Molecular Biology Joint Annual Meeting, Edmonton, June 1994.

"Occupations Associated With Breast Cancer In Alberta". **S Fincham**, A MacMillan. Accepted but not presented. Society for Epidemiological Research Miami, Florida, June 1994.

"Occupations Associated With Increased Risk for Breast Cancer In Alberta. **S Fincham**, A MacMillan. Accepted but not presented. International Symposium On Human Health and Environment, Parma, Italy, September 1994.